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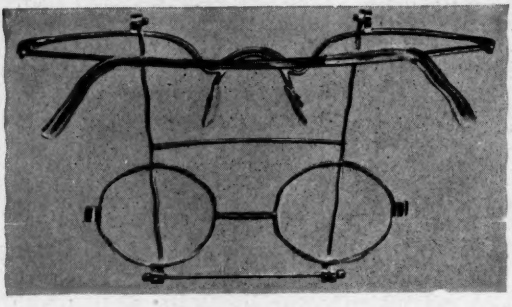

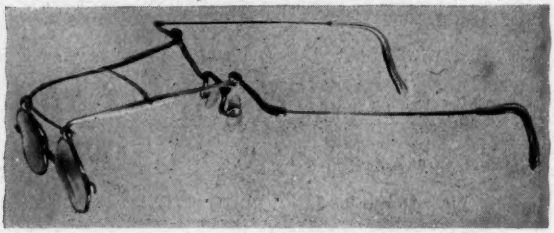

These Operating Spectacles, designed by Mr. H. B. Stallard, F.R.C.S., were the subject of an article by him in the British *Journal of Ophthalmology, October,* 1950. They present the following advantages :—

(1) When the extension carrying the lenses is swung upwards in front of the forehead, there is no obstruction below or to the sides of the operator's field of view. (2) The lenses, being placed well in front of the operator's nose and the plane of his mask, never become steamed over. (3) The spectacles are very light and comfortable, exerting the minimum of pressure on the nose and ears. (4) They can be folded flat into a small space.

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Brit. J. Ophthal. (1953) 37, 449.

COMMUNICATIONS

EDITORIAL NOTE

THE Editorial Committee feel that readers of this Journal will be interested in the following paper on Ocular Haloes and Coronas by Sir George C. Simpson, F.R.S., one of the foremost meteorologists in Great Britain, and Director of the Meteorological Office, London, 1920–1938. A late President of the Royal Meteorological Society and the recipient of their Symons Gold Medal in 1930, Simpson carried out his meteorological research in England, India, and the Antarctic. It was primarily concerned with atmospheric electricity, especially the mechanism of thunderstorms, but he was always interested in the haloes, coronas, and rainbows which are such an important feature of meteorological phenomena; this research work was published in a paper "*Coronas and Iridescent Clouds*" as a result of the observations he made while a member of Captain Scott's Antarctic Expedition, 1910-12.

In meteorology there are three kinds of such rings: the large white rings showing little colour, called haloes, which are only seen on high clouds in Great Britain and are caused by ice crystals in the air; the highly coloured coronas seen on thin clouds around the sun and moon which are due to the small water drops of which clouds are formed; and the familiar rainbow which is due to raindrops. The physics of these rings has been studied intensively, particularly by meteorologists, and Simpson was always particularly interested in them since he was able, early in his career, to prove by the "white rainbow" which he observed in the Antarctic that cloud particles can exist in the liquid state at temperatures as low as −29°C. It was natural, therefore, when in the later years of his life he began to see coloured rings around lights and was informed that they were due to particles in the eye, that he should compare them with the meteorological rings he knew so well, and study in how far they are explicable by the same physical principles.

We are indebted to his ophthalmic surgeon for the information that he first saw these coloured haloes at the end of the year 1949; although his ocular tension was normal it was presumed that he was suffering from attacks of mild congestive glaucoma and a bilateral iridectomy was performed. Although the other symptoms disappeared after the operations and the visual acuity improved, the coloured rings persisted and have been made the subject of this paper. During the last 3 years his visual fields and tension have remained normal but the lenses have shown an increase in refractive index.

His visual status in March, 1953, was:

R. vision 6/24; with −1·0 D.S., +1·5 D.C. axis 20=6/6
L. vision 6/36; with −1·75 D.S., +1·5 D.C. axis 20=6/6

Brit. J. Ophthal. (1953) 37, 450.

OCULAR HALOES AND CORONAS*

BY

G. C. SIMPSON

London

INTRODUCTION

FOR this paper to serve its purpose it is necessary that an account should be given of its origin and how it came to be written.

I am a meteorologist by profession and one of my early meteorological papers, entitled "On Coronas and Iridescent Clouds", was published in 1912. From that time onwards I have been particularly interested in the haloes and coronas seen on the clouds around the sun and moon and have made a close study of the physics of such phenomena. In January, 1950, in my 72nd year, I observed on a dark night that the street lights were surrounded by coloured rings. Thinking that these were of meteorological origin I was surprised not to be able to find anything in the state of the atmosphere to account for them. On subsequent nights the rings were still visible and as there could now be no possibility of a meteorological origin I consulted an ophthalmic surgeon. He found signs of mild congestive glaucoma and advised operations of basal iridectomies on both eyes. These operations were successfully performed in February and March and all traces of glaucoma disappeared. The coloured rings, however, continued to be seen.

By this time I was naturally very interested in the physics of the rings and having no acquaintance with ophthalmology I concluded that they were similar to the meteorological coronas with which I am so familiar. The latter are due to the small particles of water of which clouds are composed, and I jumped to the conclusion that my coronas were also caused by small particles either on the surface or within the eyes.

I soon found that the coloured rings I see are not part of a corona formed by particles, but a circular spectrum produced by the radial fibres of which the crystalline lens is built up acting as a circular optical grating. Although ophthalmologists have known this form of coloured rings, which they call the "lenticular halo", and the physics of its formation, since Druault described and explained it in 1897, I know of no reference to a circular optical grating producing a circular spectrum in any text-book of physics. I therefore continued my study of the exceptionally brilliant halo which I see. Chapter I of this paper contains a record of my study of the lenticular halo and gives a detailed account of the physics and optics of this interesting, if unimportant, phenomenon.

One dark morning in January, 1951, on looking out of a window shortly after waking, I saw a brilliant, unfamiliar corona around a near street lamp

* Received for publication March 9, 1953.

which completely swamped my lenticular halo. This time there could be no doubt that I was seeing a corona formed by particles similar to those seen on clouds; but I had no time to make any measurements before it disappeared. I did not see such a corona again until November, 1951, when I saw one around the flame of a match which I was holding in my hand, also shortly after waking. Connecting the corona with waking, I commenced what proved to be a long series of observations. For 5 months I looked for the corona each morning immediately on waking: it appeared for a few minutes on 86 out of 140 mornings and at no other time of the day. Each morning on which the corona was visible I carried out a series of observations which resulted in a great deal of information on the origin and physics of the corona and this forms the subject matter of Chapter II. This form of corona was first described by Descartes in 1637, in what must have been the first scientific description of a physiological corona. I therefore suggest that as ophthalmologists have no special name for this form of physiological corona they might adopt the name " Descartes corona".

I have already mentioned that the lenticular halo, the subject of Chapter I, is a circular spectrum produced by a circular optical grating. Now in a normal linear spectrum formed by a rectangular optical grating there is no light between the image of the source and the first-order spectrum; the space within my lenticular halo should therefore have been dark, but it was not. When I realized that there was no connexion between the coloured rings of the halo and the white light within it, it became clear that another origin had to be sought.

By questioning my friends I found that although they saw no coloured rings round a bright light they did see a glow around it, which agreed well with what I saw within my halo. I found that this glow seen by normal eyes is called the " ciliary corona " because of the large number of fine threads of light which radiate across it from the light in the centre. Chapter III is devoted to a description of the ciliary corona and the rays of light of which it is largely composed. I could find no explanation of how this glow is formed beyond "scattering of the light in the eye", but I give reasons to believe that it is a diffraction effect: namely, the aureole (the bright centre) of a corona formed by particles of a uniform size distributed at random within the eye.

Thus this paper deals with three major diffraction effects having their origin within the eye:

(*a*) the lenticular halo, due to a circular optical grating formed by the radial fibres at the periphery of the crystalline lens (Chapter I),

(*b*) the Descartes corona, due to particles on the anterior surface of the cornea (Chapter II),

(*c*) the ciliary corona with its rays, which I suggest is due to uniform particles within the eye, the exact position of which has not yet been determined (Chapter III).

I do not think that there is anything essentially new in (*a*) and (*b*); but so far as I know the ciliary corona and its bright rays have not previously been recognized as a diffraction effect. This study was not undertaken as a research; but as the duty of a physicist to make a full record of a physical phenomenon he is in a unique position to observe and investigate. If the work is found useful to ophthalmologists or physicists the credit must go to Mr. L. H. Savin who encouraged me to make the observations and gave me several opportunities to discuss them with himself and his colleagues; I should like to thank Dr. W. S. Stiles of the National Physics Laboratory for arranging the apparatus for me to see several effects which I could not see elsewhere and for his valuable advice; I wish also to thank Mr. E. F. Fincham of the Institute of Ophthalmology, London, who placed his experience of the optics of the eye, especially of the lenticular halo, at my disposal.

Chapter I

The Lenticular Halo

General Description of What is Seen.—When I observe a small source of light in an otherwise dark room there appears to be a series of coloured rings around it. Coloured rings of this nature are well known in physics and ophthalmology, there are several different kinds, each due to a different cause; as stated in the introduction we discuss three of these in this paper. In this chapter a description is given of the lenticular halo, the name given to coloured rings due to the radial fibres of which the crystalline lens of the eye is built up. Many people with normal eyes can see traces of these rings, especially when the pupil of the eye is dilated by an application of a mydriatic, but in my case the halo is so bright that I see it at night as a brilliant object around all bright lights, even in a well lighted room, and during the day around images of the sun reflected in polished surfaces. It is a striking object around the full moon, and I have seen it, but rather faintly, around a crescent moon only 4 days old.

In Fig. 1, lenticular haloes are shown diagrammatically around two similar electric street lamps, each about 25 ft high, the nearer, A, being approximately 75 ft away, and the other, B, about 100 yards away. A halo is seen around each light, but compared with the equal heights of the lamp posts the halo around B appears larger than the halo around A. This is because

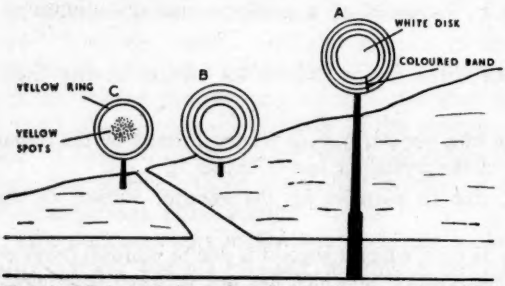

FIG. 1.—Coloured rings around street lamps. A and B, white light; C, yellow sodium light.

the haloes always subtend the same angle at the eye whatever the distance of the light around which they are centred. For this reason the halo seen around the flame of a match held at arm's length appears no larger than the length of the match stick; but it subtends the same angle as the large haloes seen around the street lamps.

The source of the light in the street lamps, shown at A and B in Fig. 1, is an electric filament which is too bright to be observed in detail. Immediately surrounding the blinding light of the filament there is a disc of light which shows no colours and decreases in brightness from the centre outwards. Concentric with the disc and in contact with it all round is the broad circular coloured band composing the lenticular halo itself. The halo is composed of coloured rings in the order of the spectrum colours, from violet near the disc to red on the outer border.

At first I thought that the central white disc and the surrounding coloured rings were all part of a corona similar to those seen on the clouds round the sun and moon, which consist of a bright white aureole at the centre surrounded by coloured rings. Further study however showed that this was not so, the white disc at the centre being no part of the surrounding coloured band and having quite a different origin. In this chapter we are dealing only with the latter.

In Fig. 1 it will be noticed that the upper part of the lamp posts is completely hidden by the halo, as though the halo were in front of the lamp post. When I hold a pencil at arm's length between the eye and the halo, it does not appear dark against the bright halo, but appears to go behind it. Similarly, if I look from a dark room through a window at a lamp outside, the halo appears to be within the room and completely hides the framework of the window spreading in front of the dark walls at each side.

This is not the case with coronas seen on clouds, for any obstacle between the cloud and the eye is seen silhouetted against both corona and cloud. The difference is explained by the fact that the light in one case is diffracted in the cloud beyond the obstacle, while in the other it is diffracted within the eye and swamps all other images on the retina.

Detailed Description of the Lenticular Halo.—The lenticular halo is not a homogeneous band of colour, but is built up of innumerable radial rays. An attempt has been made in Fig. 2 (overleaf) to represent the structure of the halo so far as it is possible in black and white. Of necessity the diagram is a negative, the dark streaks representing bright rays. The source of light is shown at the centre, surrounded by the disc of white light in which bright rays are depicted. As already stated, this white light between the source and the coloured rings has nothing to do with the halo. All that it is necessary to say about it here is that the rays in this disc are entirely separate from the rays of which the halo is composed. The rays in the halo are clear and sharp but very irregular; they vary in length and brightness and occasionally make appreciable angles with their neighbours. Each ray is a spectrum, all six colours of a spectrum being clearly seen on each. Generally the colours on each ray are at the same distances from the centre, thus

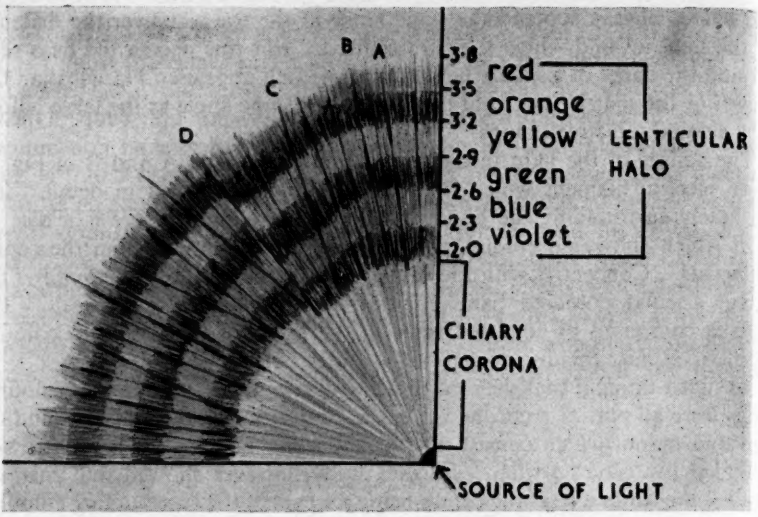

FIG. 2.—Details of coloured rings: white light.

giving rise to a series of concentric coloured rings which combine together to form the coloured band. In the diagram, alternate coloured rings have been indicated by thickening the strokes representing the rays, while the brighter rays are indicated by thickening the whole ray.

It will be noticed at once that the coloured band is not a perfect circle. The large irregularity near the middle of the quadrant occurs only in the halo seen by my left eye and is repeated in a corresponding position in the opposite quadrant. It appears as though a bundle of rays has been shortened, carrying the coloured parts inwards towards the centre. Examining the individual rays, it will be seen that the position of the colours along some of the rays are displaced, some inwards and some outwards, relatively to the general run of the colour rings. Also it will be noticed that the brighter rays extend beyond the general limit of the rays. The halo is not uniformly bright all round, some segments being appreciably brighter than others, giving rather a patchy appearance to the band as a whole.

Druault (1898) appears to have been the first to ascribe this halo to the fibres of which the crystalline lens of the eye is built acting as a circular optical grating. There is now no possible doubt that Druault's explanation is correct. A circular grating is not often met with in physics and I cannot find any such description in the text-book of physics available to me. I therefore propose to sketch the optics of a simple arrangement of a circular grating and a lens which produces a circular spectrum in all essentials similar to the lenticular halo.

Optics of a Circular Grating.—Fig. 3 shows a biconvex lens with its equatorial plane in the plane of the paper, the axis of the lens being the line through the centre perpendicular to the paper. An optical grating is shown etched on the outer zone of the lens, the lines of the grating being radial. The focal plane is parallel to the plane of the paper, but at the focal distance,

f, beyond it. The focus of the lens is at the point *F* where the axis of the lens meets the focal plane. The source of light is on the axis above the plane of the paper; it is sufficiently small and sufficiently far away for the wave front of the light from it to reach the grating in the plane of the paper so that the phase of the wave is the same at all points on the grating. The lens may be considered to consist of two parts, first the central zone within the grating, and secondly the circular zone carrying the grating. With the only source of light on the axis, all the light which passes the central zone is collected at the focus, *F*, and the focal plane therefore receives no light through the central zone except at the focus.

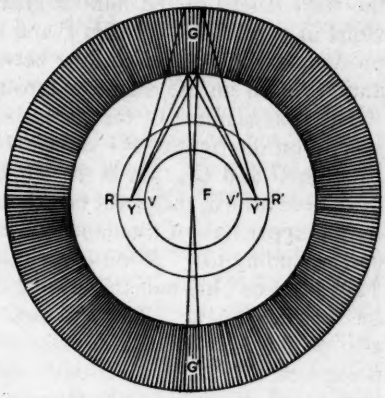

FIG. 3.—Circular grating and spectrum.

The path of the light which enters the lens through the grating can best be described by relating what happens to a beam of monochromatic light which passes through a conventional rectangular grating consisting of parallel lines engraved on a glass plate placed between a source of light and a lens. Before the grating is introduced, the lens simply forms an image of the source on the focal plane. When the grating is introduced, two more images appear, one on each side of the original image, the straight line on the focal plane joining all three images being at right angles to the lines on the grating. The distance between the central spot and the side images depends on the colour, so that when white light is used a spectrum is produced on each side of a white central spot. In this way the first order spectrum is produced; we shall not need to consider spectra of higher orders.

Returning now to our circular grating represented in Fig. 3, here again we have a grating placed between a source of light and a lens; but in this case the lines on the grating are not parallel. If, however, we consider a small segment of the circular grating between two radii near to one another, as depicted in *G* in Fig. 3, the lines in this segment are practically parallel and will act like a similar area on the rectangular grating we have already considered. Thus, when monochromatic light, say the yellow light of a sodium flame, from a source on the axis of the lens falls on *G*, it will produce three yellow images of the source on the focal plane. One will be at the main focus, *F*, where it combines with the light from the clear part of the lens, and two diffracted images at *Y* and *Y'* on a line *Y F Y'*, the diameter through *F* perpendicular to the lines of the grating in *G*. If red light is now substituted for the yellow light, red spots will be formed at *R* and *R'* on the same diameter, the distances *F R* and *F R'* being greater than *F Y* and *F Y'* because

the wave-length of red light is greater than that of yellow light; similarly violet light produces spots at V and V'. Thus white light passing through G produces two line spectra, one between V and R and the other between V' and R', both on the diameter through F perpendicular to the lines in G.

The next segment of the grating on the right of G will produce two more spectra on the right of $R\,V$ and $R'\,V'$ respectively. When all such segments between G and G', that is the whole of the right-hand half of the grating, have been added, there will be spectra corresponding to $R\,V$ on every radius on the upper half of the band between $R\,V$ and $V'\,R'$, and similarly spectra corresponding to $V'\,R'$ on every radius in the lower part of the same band. Thus it needs only half the grating to produce a complete circular spectrum on the focal plane. The left-hand half of the grating exposed alone will also produce a complete circular spectrum. Thus when the whole grating is exposed two complete circular spectra are formed on the focal plane, but as they fall on one another they combine to form a single circular spectrum.

We are now in a position to apply this simple theory of the optics of a circular grating to the problem of the formation of the lenticular halo.

Formation of the Lenticular Halo.—The eye is a complex optical system, but for this discussion, if due care is used, it may well be represented by the simple lens and grating we have used in the above simplified theoretical discussion. The bi-convex lens, the optical grating, and the focal plane will then represent the compound lens of the eye, the radial fibres of the lens, and the retina respectively, more or less in their true relative positions.

At first it is difficult to form a mental image of the fibres in the crystalline lens which form the grating. This is because most of us who have learnt our optics from elementary text-books of physics think of an optical grating as composed of a series of parallel opaque lines drawn on a transparent surface with the lines generally of the same thickness as the transparent spaces between. Consequently when one is told that the lenticular halo is formed by fibres in the crystalline lens, the fibres are thought of as opaque threads in the transparent substance of the lens. The grating with opaque lines however is only one form of optical grating, and is used in elementary text-books because it lends itself to easy numerical calculations. The strict definition of a grating has been given by Schuster (1904):

> a grating is a surface having a periodic structure which impresses a periodic alteration of phase or intensity on a transmitted or reflected wave of light . . .

Thus a grating may be formed by laying fine straight glass fibres side by side and touching one another, the "periodic alteration of phase" being set up by the shape of the glass fibres. The fibres in the lens of the eye are of this nature; without being too technical, they may be described as long strips of transparent material, having a cross section of hexagonal

FIG. 4.—Lens fibres.

shape, but with two opposite sides of the hexagon much longer than the other, packed tightly together as in Fig. 4. A structure of this kind makes a very effective grating while being quite transparent.

The whole substance of the crystalline lens is composed of fibres of this nature; their diffractive effect is however limited to the periphery, where, on the whole, they are uniform in size and radial in direction, but there are local irregularities which we shall have to study later. The central part of the lens, although built up of similar fibres, shows no diffraction and therefore may be considered to be optically homogeneous.

The focal plane of the theoretical discussion corresponds to the retina in the eye. With a perfect circular grating on the crystalline lens there would be a perfect circular spectrum formed on the retina. When the eye "looks at" a point source of light at its own level, the axis of the eye is horizontal, the plane of the retina is vertical, and the light from the source is brought to a focus where the axis meets the retina. We have now on the vertical plane of the retina a physical point of white light surrounded by a physical band of rings of coloured light corresponding to the circular spectrum in Fig. 3. Converted into sensation, this produces the appearance of a halo with perfect rings of coloured light on a vertical plane surrounding the source of light. But the lenticular halo is not composed of perfect rings of coloured light; it is composed of coloured rays of unequal brightness and unequal length, and of rings of coloured light which are far from being perfect circles. We have now to study how the structure of the halo depends on the fibrous structure of the crystalline lens.

In the theoretical discussion, we found that the spectrum on the radius at *R V* (Fig. 3), is due to the lines in the two small segments of the grating at *G* and *G'* combined, and that the spectra at the other positions are also due to the lines in similar small segments of the grating. Now the distance of any colour in a spectrum from the image of the source (the deviation) depends on the distance between corresponding points on adjacent lines in the grating (*i.e.*, in our case, on the distance between the centres of adjacent fibres). Hence when the colours on a ray in the halo are displaced towards the centre relatively to the general run of the rings, for example the ray at *A* in Fig. 2, we know that the fibres which cause this ray are wider than the average fibre. Similarly if the colours on another ray are displaced away from the centre, as at *B* in Fig. 2, we know that this is caused by fibres below the average in width. We shall later see how to determine where the fibres causing any ray are situated, but at present it is sufficient to know that the irregularities of the colour band indicate variations in the widths of the fibres in different positions on the outer zone of the crystalline lens. It is not certain why the rays vary so much in brightness; it is not likely to be due to the crowding of the fibres together, for that would involve reducing the width of the lines when all the brighter rays would have longer spectra which they have not; it is more likely to be due to bunches of fibres which

are more uniform both in size and direction than usual, for example like a tress of hair which has been straightened by slight tension, the diffraction of such a bundle would be more perfect than of a less regular bunch of fibres and therefore brighter. At *C* there is a ray which is far from being radial; this must be due to a bunch of fibres being pulled out of place so that their direction is not truly radial. Between *D* and *C* the rings are abnormally disturbed, a large section being bodily displaced towards the centre, and this is repeated in the opposite quadrant. The natural explanation is that an appreciable section of the fibres in the left eye (there is no similar irregularity in the halo of the right eye) are abnormally wide, involving contracted spectra. It is interesting and instructive to locate the position of this abnormality in the fibres of the crystalline lens of the eye. In drawing Fig. 2, the source of light was supposed to be at the same height as the eye, hence the axis of the lens was horizontal and the plane of the retina and the apparent plane of the halo were in consequence both vertical. The halo reproduces the image on the retina; but we know that the image on the retina is inverted, *i.e.*, rotated through 180°, relatively to the object of which it is the image. Thus the top and left side of the halo correspond with the bottom and right side respectively of the image on the retina. Hence the image of the distortion in the top left-hand quadrant of the halo in Fig. 2 must be in the bottom right-hand quadrant of the image on the retina in Fig. 3. The rays in the image on the retina, however, are produced by fibres in the grating which are perpendicular to the rays, hence the rays in the bottom right-hand quadrant of Fig. 3 are produced by the fibres in both the top right-hand quadrant and the bottom left-hand quadrant of the grating. Thus the abnormal fibres may be in either of these two quadrants, but in which we cannot say without further data. We know, however, that if one half of the pupil is covered we still see a complete halo, formed by the half of the grating still uncovered. Thus if we cover each half of the pupil in turn we shall see the defect in the halo only when that half of the grating in which the defect is situated is uncovered. In the case we are discussing, it was observed that the defect could be seen when the right-hand side of the eye was uncovered but not when it was covered. It is clear therefore that the defect is in the top right-hand quadrant of the grating. In this way I have found no difficulty in locating on the grating the position of any irregularity which gives rise to an outstanding ray or marked distortion in the lenticular halo of either of my eyes. This result is very important, not for its practical value in locating the position of a defect, but because it proves conclusively that the rays in the lenticular halo are formed by irregularities in the spacing of the fibres in the crystalline lens.

Size of the Lenticular Halo.—The size of a halo can only be expressed in angular measure. If there is a small source of light (say a hole with a lamp behind) on an extensive vertical screen at the height of the eye, the halo always appears to be on the screen. It is then a simple matter to mark the

position on the screen where any part of the halo appears to be situated. Let us in this way mark the positions of the end of the horizontal radius of any coloured ring of the halo and measure its distance from the centre, say x cm. If at the same time we measure the distance of the eye from the screen and find it y cm., then the ratio x/y is sin θ, in which θ is the angle subtended at the eye by the radius of the ring measured. It is this angle θ which is usually given as the " size " of the rings of a halo or corona, for it is constant and also independent of the distance of the source of light. There are several practical ways of measuring θ; the method I used is described in detail in the Appendix.

Physics of an Optical Grating.—We have seen in the paragraphs dealing with the optics of a circular grating that, although the lines on a circular grating are not strictly parallel, the number involved in producing the spectrum on any radius of the halo is so small that we may consider the lines parallel and of constant thickness. Thus the laws of a normal rectangular grating can be applied to the lenticular circular grating. The fundamental equation of a grating is:

$$\sin \theta = \lambda/e \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (1)$$

in which θ is the deviation from the direct path produced by the grating on light of wavelength λ (it is therefore in our case the angle subtended at the eye by the radius of the ring in the halo formed by light of wavelength λ),

and e is the distance between the centres of adjacent grating spaces, *i.e.*, the width of the fibres.

As sin $\theta = x/y$, we have only to measure x and y as described for any one of the coloured rings in the halo to find the value of θ. We can then by the application of Equation (1) find the value of e.

Results of Measurements of θ.—I can detect no difference in the size of the haloes in my two eyes, and it is distinctly easier to make measurements using both eyes. The measurements shown in Table I, unless otherwise

TABLE I

MEASUREMENT OF LENTICULAR HALO

1	2	3	4	5	
Radius Measured	$\frac{x}{y}$ sin θ	Angular Radius θ (degrees)	Wavelength λ (mm.)	e (mm.)	
			$\times 10^{-5}$	$\times 10^{-4}$	
Outer limit of Red	0·068	3·9	70	97	
Orange-Yellow	0·057	3·3	59	97	mean 94
Yellow-Green	0·051	2·9	55	93	
Green-Blue	0·046	2·6	49	94	
Inner limit of Violet	0·036	1·9	40	90	

stated, were made using both eyes simultaneously. In measuring the rings, it is easier and more accurate to measure where the light changes from one colour to the next, say from yellow to green, than to measure the centre of each colour separately. In column 3 of Table I is given the value of the angular radius, θ, for each of the five positions on the halo specified in column 1, and column 4 contains the corresponding wavelengths. In column 5 are given values of e derived from θ and λ by Equation (1). Theoretically each measurement should have given the same value for e: considering that the limits of the inner violet and the outer red are not definite, the measurements are sufficiently in agreement for the mean value of e (0·0094 mm.) to be accepted as a near approximation. The actual width of the fibres from these measurements will be considered in the next paragraph. For comparison with other measurements it is convenient to know the mean angular radii for each coloured ring; this is given in Table II based on the measurements in Table I.

TABLE II

RADII OF COLOURED RINGS OF LENTICULAR HALO

Colour	Radius θ of middle of ring (degrees)	sin θ	Wavelength λ (mm.)
			$\times 10^{-5}$
Red	3·7	0·064	68
Orange	3·4	0·059	63
Yellow	3·1	0·054	57
Green	2·8	0·049	52
Blue	2·5	0·044	47
Violet	2·2	0·038	40

Size and Position of Fibres.—Equation (1) assumes that the wave front is parallel to the plane of the grating where the two meet; in other words, the incident light on entering the grating forms part of a " parallel beam." This would be true if the grating were on the surface of the cornea, but it is situated on or in the crystalline lens, the equatorial plane of which is between 5 and 6 mm. behind the cornea. The incident light strikes the cornea as a parallel beam, but the cornea acts as the first surface of a compound lens, in consequence of which the parallel beam is changed into a converging beam. It is this converging beam which strikes the grating formed by the fibres of the crystalline lens. Druault (1923, p. 490; 1950, p. 340) has calculated the effect of this and shown that if e is the apparent grating space obtained by using Equation (1), the true value of e would be $e/1{\cdot}14$ if the active fibres were on the front surface of the crystalline lens, or $e/1{\cdot}32$ if they were on the posterior surface. As we do not know exactly where the active fibres are situated, he takes the mean of these two values and divides e obtained from Equation (1) by 1·23 to obtain the true width of the fibres. The factors 1·14, 1·23, and 1·32 are called by Druault the " coefficient of position " at the anterior, equatorial, and posterior surfaces of the crystalline lens.

Applying 1·23 as the coefficient of position appropriate to the fibres of the crystalline lens to the value of e found from equation (1), namely 0·0094 mm., we find the true value of the grating space to be $0·0094/1·23 = 0·0076$ mm. We have taken the grating space to be the width of the fibres, hence in round figures, and that is all we are justified in using, the " width of the fibres " is approximately 8μ. Salzmann (1912, p. 172) gives the width of the fibres by dissection "perpendicular to the length and parallel to the surface" as $8–12\mu$.

In our discussion we have assumed that the fibres which cause the halo are confined to the outer zone of the lens and that there is no diffraction in the central part of the lens. This assumption is necessary, for if the fibres continued radially towards the centre there would be such a large difference between the grating space at the centre and at the periphery that narrow coloured rings such as are observed could not be formed. A simple direct observation however shows that this assumption is correct. If I place a card with a pinhole in it as close as possible to the pupil of the eye when observing a halo, I find that when the pinhole is near the edge of the pupil a portion of the halo appears; but when the pinhole is over the centre of the eye no sign of the halo can be seen although the source of light is clearly visible. The explanation is obvious: the pinhole confines the light from the source to a narrow beam, little larger in cross-section than the pinhole itself. When the pinhole is near the edge of the pupil the whole of this narrow beam passes through a small area of the grating, such as G in Fig. 3, and only the small sector of the halo corresponding to G can be seen. When the pinhole is over the centre of the pupil the absence of any portion of the halo indicates that the fibres do not produce diffraction there. With elaborate apparatus it might be possible by this method to mark out exactly how near to the centre the fibres cause diffraction; but there is a much simpler method which will serve our purpose.

A hole was pierced in each of three pieces of card having diameters of 2, 3, and 4 mm. respectively. Each card in succession was then held over the eye, with the hole as nearly over the centre of the pupil as possible, while a small source of light two or three metres away was viewed through the hole. The following results were observed:

(*a*) when the 2-mm. hole was used the source of light could always be seen without a halo,

(*b*) with the 3-mm. hole the card could not be held centrally for any length of time without the halo appearing faintly,

(*c*) with the 4-mm. hole the halo was always visible.

In short, a beam which is less than 3 mm. in diameter before entering the pupil can pass through the clear part of the lens without touching the grating, while a beam greater than 3 mm. in diameter before entering the pupil cannot pass through the clear space without passing through some portion of the grating also.

If it were not for the curved cornea between the hole and the grating we could have said at once that the diameter of the clear part of the crystalline lens is 3 mm.; but the beam which is parallel when it passes through the hole is converging after

passing the cornea, hence its cross section is less on passing through the crystalline lens than when it passes through the hole. Using Druault's "coefficient of position," the diameter of the clear part of the crystalline lens would be 3/1·23= 2·4 mm. These values however cannot be exact and almost certainly vary from person to person. It is sufficient for all practical purposes to say that the diameter of the clear space within the grating in my eye is approximately 2·5 mm., *i.e.* the optically active fibres extend to within 1·25 mm. of the centre of my crystalline lens. As the diameter of the pupil varies from approximately 3 mm. in daylight to 6 mm. in artificial light, the width of the grating exposed in my eye varies from less than 0·25 mm. to more than 2 mm. according to the light.

Variations in the Size of the Halo from Time to Time

I commenced systematic observations on the lenticular halo on February 10, 1950. I cannot remember how bright the rings appeared when I first saw them, but they were obviously sufficiently striking for me to take them seriously, otherwise I should not have consulted a specialist. I can, however, remember that I had no difficulty in making measurements on them and I am not conscious of any change in the brightness of the haloes since I began to keep them under observation. When glaucoma had been diagnosed, but before the operation, I underwent a course of pilocarpine drops, which were introduced into the eyes three times a day. On each application of the drops the intensity of the haloes was much reduced and this was taken to confirm the diagnosis of glaucoma. When, however, the haloes were still visible after the operation it was realized that this temporary reduction in the intensity of the halo had been due to the reduction in the amount of light entering the eye owing to the contraction of the pupil caused by the drops. In 4 or 5 hours after the application of the drops the pupils had returned to their normal size and the haloes had regained their usual brightness.

I have measured the radius of the red ring (middle of the orange and red rings) very many times since February, 1950, and especially during the 20 days before the operation, when the pilocarpine drops were being applied daily and I have never found a measurable variation in their size, not even when they were almost invisible after the application of the drops. This is what would be expected if the rings are due to the lens fibres; for these are permanent structures in the eye and their size is not likely to change, except for the slow change due to normal growth.

Tests for diagnosing the Lenticular Halo

I propose to close this account of the lenticular halo by describing the tests which have been proposed for differentiating lenticular haloes from other coloured rings seen around the lights; not so much for their practical use, but as illustrating in a striking way the chief characteristics of this halo as described above.

(*1*) *Druault's Test.*—This was described in 1898 ; a detailed account will not be given here as it is readily accessible, and having now been superseded is of only academic interest.

(*2*) *Emsley-Fincham Test.*—This was described in 1922; it is essentially the same as Druault's test, but in a greatly improved form. A screen with a slit about a millimetre wide is passed across the eye; the resulting changes in the appearance of the haloes are striking, and are spread out over the whole traverse of the slit across the pupil. In Fig. 5, which is based on Fig. 3, only the portion of the

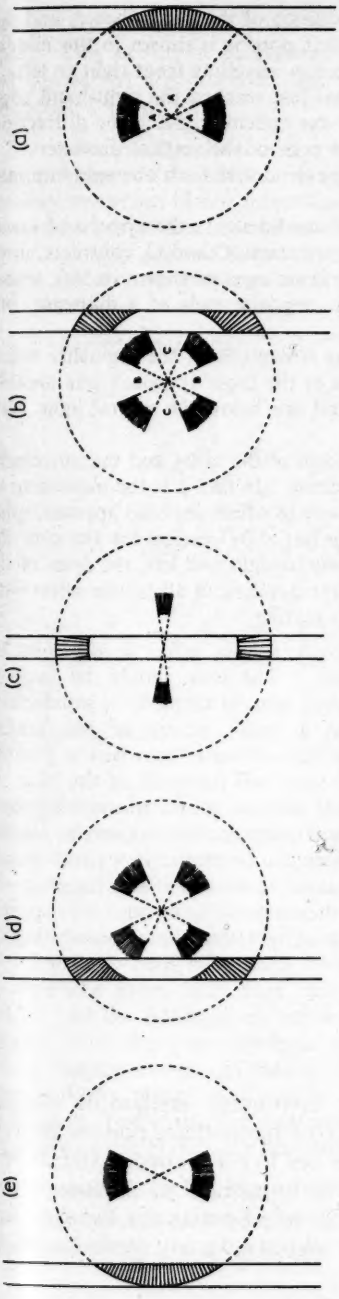

FIG. 5.—Appearances in Emsley-Fincham Test.

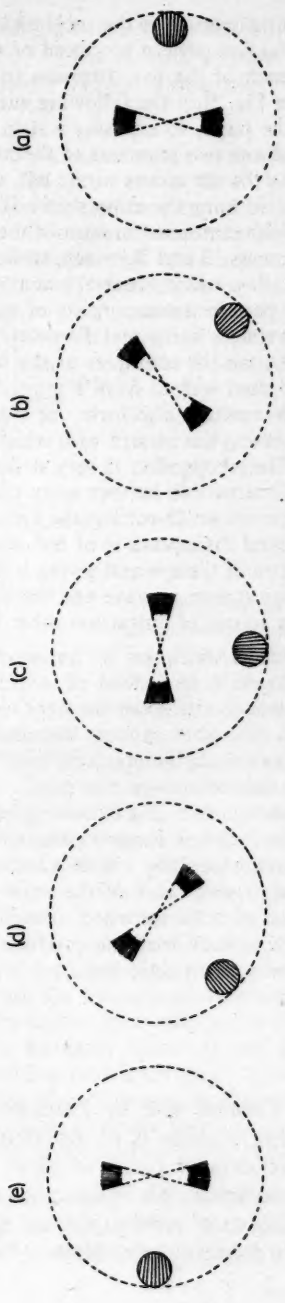

FIG. 6.—Appearances in modified version of Emsley-Fincham Test.

grating exposed to the incident light in each position of the slit is shown, and the diffraction pattern produced on the retina by that portion is shown in the middle of each of the five diagrams (*a–e*). The screen is travelling from right to left.

In Fig. 5(*a*), the following edge of the slit has just reached the right-hand edge of the pupil, so exposing a strip of grating on the extreme right. The diffraction produces two segments of the halo at opposite ends of the vertical diameter.

As the slit moves to the left, these segments extend and each one splits up into two, forming the cross shown in 5(*b*).

With continued motion of the slit to the left the breaks in the upper and lower segments, B and B, widen, while the gap between them, C and C, contracts, until the slit is exactly central when the arms of the cross meet as shown in 5(*c*), where the pattern again consists of two segments at opposite ends of a diameter, but this time a horizontal diameter.

As the slit continues to the left, the process is continued: the segments widen and then split to form a cross, 5(*d*); the arms of the cross approach one another and meet again to form one segment above and one below the central light, 5(*e*), repeating the pattern with which we started.

The whole effect is very striking, the formation of the cross and the movement of its arms can be seen when the halo is very faint. In fact it is the movement of the arms which catches the eye and the exact way in which the halo appears, splits up and disappears is of no importance in the test. By placing the slit over the centre of the eye and giving it slight movements to right and left, the arms of the cross appear to wave and there is nothing corresponding at all to this effect with any source of diffraction other than a circular grating.

(*3*) *Modification of Emsley-Fincham Test.*—A similar effect is obtained by using a hole instead of a slit in the screen. The hole should be large so as not to cut down the light too much, a hole 2 mm. in diameter is satisfactory. As this hole cannot illuminate more than a small section of the grating at any time, the complete halo can never be seen whatever may be the position of the hole over the pupil. Instead, one sees two segments of the halo, at opposite ends of a diameter (Fig. 6, p. 463). If the hole is now moved irregularly over the pupil, the two coloured spots will appear to dance and to chase one another around the light. With a little practice the hole can be moved in a circle around the circumference of the pupil when the coloured spots will rotate together in a kind of catherine wheel around the light at the centre (Fig. 6*a–e*). If a patient after a little trial can produce this effect it is certain that the coloured rings he sees is a lenticular halo.

CHAPTER II

CORONA

Coronas due to Particles.—The most convenient method of studying these coronas is to distribute a thin layer of lycopodium powder between two circular pieces of glass, which fit the eye like a monocle, and observe a distant source of light through the disc thus formed. Lycopodium powder consists of nearly spherical spores which are all of practically the same size, the diameter being about 0·C09 mm. (9μ). When a distant, small. but bright,

source of white light is viewed through such a disc, a brilliant corona will be seen. This is shown diagrammatically in Fig. 7. The corona consists of two parts (*a*) the aureole, and (*b*) the coloured bands; these must be described separately.

The Aureole.—The aureole is a bright disc of light, white at the centre and coloured towards the border. It is brightest near the centre and if the particles are very numerous blends into the source itself. The aureole decreases in brightness and tends to lose its whiteness as the distance from the centre increases: at about half-way across a tinge of yellow appears which becomes more and more red as the distance increases until the aureole ends in a broad, brown-red ring surrounded by a narrow dark ring. Thus the aureole is a bright disc of light, white near the centre, with a strongly coloured red border which forms the "first red ring" of the corona.

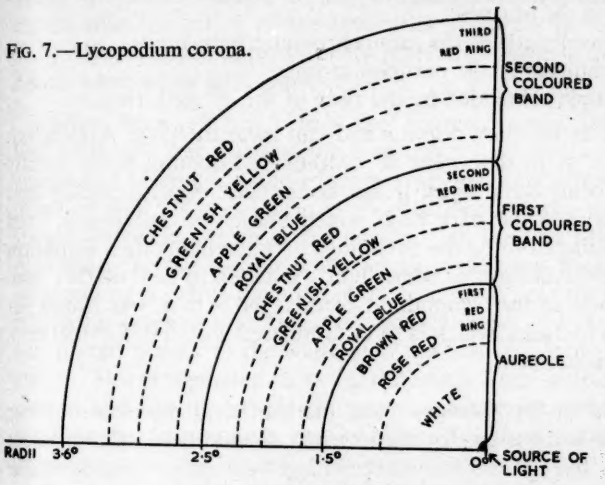

FIG. 7.—Lycopodium corona.

The Coloured Bands.—Surrounding the aureole with its red border is a series of coloured bands extending with decreasing brightness to a distance depending on the intensity of the source of light. Each band consists of a series of coloured rings, the colours (proceeding outwards) being blue, green, yellow, and red. This is the order of the colours in the spectrum, but these are not spectrum colours but "subtraction colours," for they result from subtracting spectrum colours from white light. The first ring is blue because yellow and red have been subtracted, the second green because red and blue have been subtracted, and so on to the final red ring from which the blue and green have been subtracted; they are the colours seen in soap bubbles and the pools of oil left on wet roads by motor cars; they are quite different from the pure spectrum colours of the lenticular halo. It will be noticed that the aureole and each of the coloured bands ends in a red ring. These red rings have a sharp junction with the dark blue ring of the following band which makes them good marks for measurement, and they have generally been used for this purpose since Fraunhofer first measured their radii in 1820 (Fraunhofer, 1823).

Measurement of the Size of the Particles forming the Corona.—The mathematical treatment of the formation of the corona has only been worked out for monochromatic light. With monochromatic light the

corona consists of the aureole, surrounded by a number of bright rings with dark rings between them. The aureole itself is the first bright ring and therefore the first dark ring is the boundary of the aureole; bright and dark rings then follow in succession.

Formulae have been given relating the angle subtended at the eye by the radii of the rings, the diameter of the particles, and the wavelength of the monochromatic light. For the dark rings the formula is:

$$\sin \theta_n = \frac{n + 0{\cdot}22\lambda}{d} \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (2)$$

in which n is the number of the dark ring ($n=1$ for the dark ring bounding the aureole);
λ is the wavelength of the monochromatic light used;
d is the diameter of the particles in mm.;
θ_n is the angle subtended by the radii of the n^{th} dark ring.

It will be seen that, as we know λ and n and can measure θ (see Appendix), Equation (2) allows us to determine d. Although Equation (2) is strictly true only for monochromatic light, it has been found experimentally that when white light is used the dark rings which border the successive " red rings " may be considered to be the dark rings of white light; then Equation (2) holds if λ, " the wavelength of white light", is taken to be 0·000571 mm. Applying this method to the lycopodium corona, 0·029 mm. was found for the diameter of the particles, which is to be compared with 0·021–0·030 mm. found by direct measurement.

Intensity of Light in the Corona.—The mathematical discussion of the corona also leads to expressions for the intensity of the light in the different parts of the corona, but again for monochromatic light only. The formulae show that the intensity of the light (of all wavelengths), is greatest in the centre of the aureole near to the source of light; from this high intensity as one proceeds from the centre outwards the intensity decreases, slowly at first and then very rapidly to zero at the limit of the aureole; the intensity then rises to the maximum of the first ring and then there is a series of maxima with rings of no light (minima) between them. The ratio of the light from maximum to maximum is the same for all wavelengths, so that Table III applies to all wavelengths. From this it will be seen how much brighter the aureole is than the coloured rings. Yet it is possible to see four or five coloured bands with a good lycopodium disc and a bright light.

These intensities are the theoretical intensities for monochromatic light; now the brightness of colour is not the same thing as the intensity of light, therefore we are not justified in adding the intensities of several monochromatic lights to obtain the brightness of the mixture. We have however found that treating white light as monochromatic light of wavelength 0·000571 mm. gave useful results in determining the size of the particles forming the corona; we shall, therefore, in the absence of other means,

TABLE III
INTENSITY OF LIGHT IN A CORONA DUE TO PARTICLES

Zone	Intensity
At centre of aureole	1,000
One-quarter across aureole	800
Half-way across aureole	360
Three-quarters across aureole	80
At circumference of aureole	0
At brightest part of first coloured band	17
At brightest part of second coloured band	4
At brightest part of third coloured band	2

apply the same method to the problem of brightness. Hence we may expect that the ratios of the intensities given for monochromatic light in Table III will apply, approximately at least, to the brightness of the coloured bands when white light is used.

Corona Seen.—When I first saw coloured rings around street lights I had no doubt that I was seeing a corona similar to the one I have just described and which I had studied many years previously from the meteorological point of view. I was however, puzzled, because the rings I saw varied in several particulars from a true corona. In the first place, the central disc of white light was distinctly less bright in all parts than the coloured band surrounding it, while the aureole of the corona near the centre is more than fifty times brighter than the first coloured band. In the second place there was no red border to the white disc, the first coloured ring being distinctly violet. It was suggested to me that the rings I saw were due to the lenticular grating and not to particles in the eye; the application of the Emsley and Fincham test immediately proved this to be the case and I commenced the investigation of the lenticular halo described in Chapter I.

One morning in January, 1951, I looked out of the window while it was still dark and the street lamps were still alight, and then I saw around the nearest street lamp not the halo with which I had by then become very familiar, but a corona with a brilliant aureole and two conspicuous red rings. There could be no doubt I was at last seeing a real corona due to particles. The corona disappeared before I had time to make any measurements and was replaced by the lenticular halo in its usual form. I did not see a repetition of the corona for nearly a year, and then in November, 1951, I recognized it again, this time around the flame of a match. On both occasions I saw it early in the morning within a few minutes of waking. I therefore decided to look for it each morning immediately after waking and saw it on nine of the next twenty mornings but not at any other time of the day. I was now quite sure that I was seeing a true corona due to particles, something quite different from the halo which I can see at all times of the day and night. It was then November and therefore dark when I woke up each morning; so I fixed up in my study, next door to my bedroom, a convenient point source of light and the necessary apparatus for measuring the radii of the rings seen around it.

Method used in Observations of the Corona.—The following procedure was developed after a few days of experience. I was generally asleep when the alarm woke me at 6·48 a.m., and I immediately went into the study. This took one or two minutes during which I did not use my eyes more than was necessary to see my way about—I kept them closed most of the time. On arriving in the study I switched on the room light, then after a rapid look round to see that the apparatus was in order I switched it off again, turned on the experimental source of light, and stood in front of it to make the observations planned for that morning.

Results of Observations.—I rediscovered the morning corona on November 7, 1951, and observations were made on nearly every subsequent morning until March 25, 1952, when the increasing morning light (I had no completely dark room) put an end to them.

The Liquid Glare.—On first looking at the bright spot of light, within one or two minutes of waking, it was generally seen to be distorted by a film of moisture over the eye. This film was practically always present, sometimes more and sometimes less; its presence could be recognized by bright and dark areas with bright outlines in the field of view, just as one sees in the shadow of a sheet of glass on which a little water has been spilt. I call this effect " liquid glare." I have no reason to believe that the moisture in my eyes was excessive, there was no " weeping " and no deposits in the corners of the eyes; it was the normal moisture which accumulates under the eye-lids during sleep.

Unless the moisture was unusually pronounced, the normal lenticular halo could always be seen through the liquid glare for the first few minutes of observation; but there was no sign of any corona. During 5 months of daily observations there was only one morning on which a corona was present when I first looked at the source of light and on that morning the eye was inflamed by a stye. Normally it takes between 2 and 5 minutes from waking for a corona to appear. The importance of this interval will be discussed later.

If while continually watching the background around the central bright light—frequently covering first one eye then the other with the hollow of the hand in order to compare the development in the two eyes—a corona did not develop, the liquid glare slowly disappeared and the lenticular halo became clearer until it had regained its normal appearance. The observation was then over for that morning, for if the corona did not appear within 5 or 6 minutes of waking it was not seen that day.

Development.—If the corona is going to develop, the background within the lenticular halo becomes brighter and the outer red ring of the halo appears to become stronger and broader. This however is not a change in the halo, but the first sign of the formation of the aureole of the corona, the bright centre and red border of which cover the halo and swamp it. A green band then begins to appear outside the red border of the aureole. As the green band gets brighter, the second red ring appears beyond it, and finally we have a trace of green light beyond the second red ring. I have never seen the third red ring with certainty; but I have thought I got glimpses of it, no doubt if my light had been stronger I should have seen a third red ring.

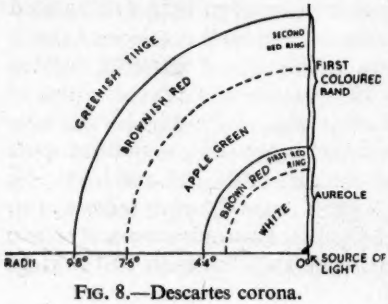

FIG. 8.—Descartes corona.

The fully developed corona which I see consists of a bright central disc surrounded by three coloured rings, the inner and outer being brownish-red and the one between apple-green. The corona is shown diagrammatically in Fig. 8.

Disappearance.—The corona does not remain visible for many minutes and has

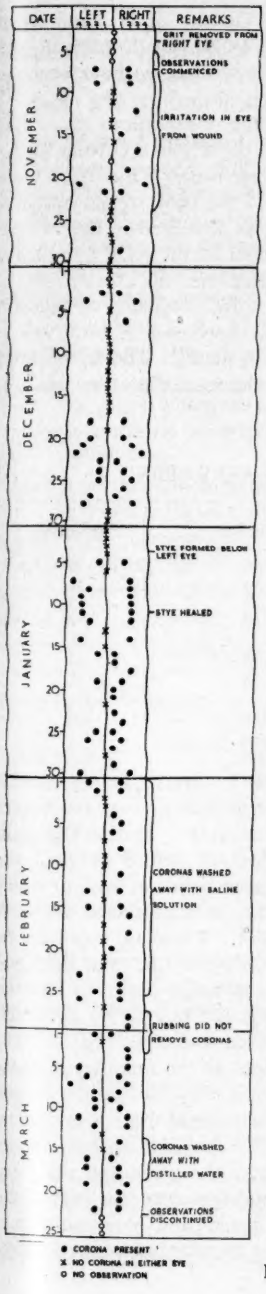

FIG. 9.—Frequency of Descartes coronas.

generally disappeared 15 minutes after waking. The disappearance is not sudden: the first sign that the corona has started to weaken is that I can see the halo through the corona; then the rings of the corona grow fainter and the halo by contrast clearer and sharper.

It has been stated in the literature that coronas disappear on blinking or on rubbing the eye. On twenty occasions when a corona was clearly visible I made tests to find the effect of blinking, rubbing, and washing the eye with distilled water or saline solution in an eye bath. There could be no possible doubt that neither blinking nor rubbing removed the corona: in fact, blinking appeared to accelerate its appearance, and although rubbing reduced the brightness of the corona at first, it soon recovered. On the other hand, washing the eye with distilled water or a saline solution removed the corona immediately. These experiments leave little doubt that the particles causing the corona are not within the eye, but on the surface of the cornea.

Frequency with which Coronas were seen.—I never saw a corona except in the morning within a few minutes of waking from sleep. They occurred in either eye, in both eyes simultaneously, or in neither. They occurred with large variations in intensity from day to day, from slight tinges of colour through which the lenticular halo could still be seen, to a brilliance which caused me one morning to enter in my notes "on using both eyes simultaneously the corona was glorious, there is no other word for it." (The bare filament of a 40-watt lamp seen through a 4-mm. diameter hole was used in this observation.) In order to record these variations I adopted a rough scale of intensity from 0 to 4 which was entirely subjective, and therefore cannot be used quantitatively for comparing individual observations, but which served a qualitative purpose.

Observations were made on 140 mornings and the results are shown graphically in Fig. 9. In this diagram the intensity of the corona, according to the scale mentioned above, is shown by a dot, to the left of the zero line for the left eye, and to the right of it for the right eye; a cross on the zero line indicates that there was no corona in either eye, and a circle that no observation was made that day.

Coronas were seen in one or both eyes on 61 per cent. of the morning observations, but they were very irregularly distributed. In December there was a sequence of 11 days during which coronas were not seen in either eye, while in March there was a

sequence of 10 days with coronas visible every day. On the other hand there was a period of 18 days (February 6–23) during which a corona appeared in one or both eyes on almost alternate days and no sequence with or without coronas lasted for more than 2 days.

Taking the two eyes separately: in 39 per cent. of the observations there was no corona in either eye; of the remaining 61 per cent. there was a corona in the right eye in every case, but in the left eye in only 36 per cent. Thus there were coronas in the right eye without one in the left eye in 25 per cent. of the observations; but there was not a single case in which a corona appeared in the left eye without one in the right eye. Because coronas appeared in the right eye nearly twice as often as in the left eye it must not be concluded that the coronas in the two eyes were independent of each other, for there was a high degree of correlation between the intensities in the two eyes when coronas appeared in both simultaneously, as will be clearly seen if Fig. 9 is examined in detail. The significance of these statistical results will be considered later, in the meantime they are collected together in Table IV.

TABLE IV
FREQUENCY WITH WHICH CORONAS WERE SEEN

Site	Number	Percentage
Neither eye	54	39
Right and left eyes simultaneously	51	36
Right eye only	35	25
Left eye only	0	0
Total mornings with corona	86	61
Total mornings without corona	54	39
Grand Total	140	100

Colours of the Corona.—The typical corona which I observe is represented in Fig. 8. It consists, as already stated, of a white aureole with a broad red border followed by a broader green ring and then by another red ring. This, at first sight appears to differ from the corona produced by lycopodium powder (Fig. 7), the aureole of which is surrounded by a sequence of coloured bands in each of which blue, green, yellow, and red rings can be recognized in accordance with the theoretical explanation of coronas formed by particles. There is, however, no real difference. The theoretical discussion deals with a point source of light and equal particles. When the particles are of the same average size, but with an appreciable range of individual sizes, the coloured rings are broadened and overlapping takes place. Consider now the first coloured band in Fig. 7. The intensity of the rings in this band is three times as great as the intensity of those in the next band, therefore we may neglect the latter, and consider that the corona ends with the second red ring. When the red ring is broadened there is no mixing on the outer edge, therefore the ring will be broadened and remain red; on the other hand, the other rings will overlap, the yellow with the green and the green with the blue. All these mixtures have a green hue and therefore this part of the coloured band will lose its clear cut rings of yellow, green, and blue and appear as a green band, slightly blue on the inside and blending into the red on the outside.

These small differences of tint are hardly perceptible and the appearance is as described: an apple-green band between two red bands surrounding the white centre of the aureole. From this we learn that the particles forming the coronas are much less uniform in size than the spores of lycopodium.

Size of the Corona.—From what has been said it is clear that the red rings in the observed corona, Fig. 8, correspond with the red rings in the theoretical corona, Fig. 7. We can therefore determine the size of the particles on the cornea which cause the coronas by measuring the outer radii of the two red rings.

It was not easy to make this measurement, for the coronas are so transient that by the time one was sure that the corona had developed sufficiently for a measurement to be made, there was very little time left for making the measurement. In all, I was able to make 83 separate measurements on 14 days, and from these it is possible to say that:

(*a*) the size of the rings was remarkably constant, the variations from day to day being well within the somewhat wide limits of observational error;

(*b*) there was no consistent change in the size during the measurements on any one occasion.

From (*a*) we can conclude that the average size of the particles was the same from day to day, and from (*b*) that they did not grow in size after appearing nor decrease in size before disappearing.

Table V, giving the dimensions of each ring of the corona, has been constructed from the means of all the observations.

TABLE V

RADII OF COLOURED RINGS OF CORONAS

Ring or Band	Position of Measurement	sin θ	θ
First red ring	Inner edge (approx.)...	0·057	3·3
	Brightest part...	0·071	4·1
	Outer edge Inner edge }	0·077	4·4
Green band...	Middle	—	(6·0)
	Outer edge Inner edge }	0·132	7·6
Second red ring	Mean radius	0·144	8·6
	Boundary (approx.)	0·170	9·8

We can now find the size of the particles responsible for the coronas by putting the values for sin θ for the two red rings from Table V in equation (2), and taking $\lambda = 0{\cdot}000571$ (the wavelength of white light).

First red ring, $n = 1$, $d = \frac{1{\cdot}22}{0{\cdot}077} \times 5{\cdot}71 \times 10^{-4} = 0{\cdot}0091$ mm.

Second red ring, $n = 2$, $d = \frac{2{\cdot}22}{0{\cdot}17} \times 5{\cdot}71 \times 10^{-4} = 0{\cdot}0074$ mm.

These two values for d should be the same; the measurements, however, were too consistent for the discrepancy to be due to observational error; it is much more likely to be due to the large spread in the size of the particles. In any case the difference is of little importance, and the mean of the two, 0·008 mm. (8μ), cannot

be far from the average diameter of the particles. For comparison we may note that the diameters of the lycopodium spores, cloud particles, and red blood corpuscles are 21 to 30μ, 4 to 20μ, and 7 to 8μ respectively, while the limit of unaided vision may be taken to be 50μ.

Rays.—In common with other coronas due to particles, morning coronas, under certain conditions, take on a radial structure. The rays are well marked in the aureole and in the first red ring and can be seen to half-way across the green band. The difference between these rays and those of the lenticular halo is that the halo is composed entirely of rays which can be seen individually, while in the corona the rays appear to be superimposed upon the aureole and coloured rings, which otherwise are uniform. These rays will be further discussed in Chapter III.

Nature of the Particles causing the Coronas

These morning coronas have long been recognized for what they are: the diffraction effect of particles within or upon the surface of the eye. But the problem of what the particles are is far from solved. I will give quotations going back for nearly a century in which suggestions are made on the nature of the particles.

(*a*) Les couronnes qu'on voit souvent, autour des chandelles, le matin en se levant, sont dues à des globules de sang injectés dans la conjonctive (*sic*) pendant le sommeil. Billet (1858).

(*b*) Il est sans doute produit par des globules de pus étalés à la surface de la cornée. Druault (1899, p. 13).

(*c*) For a period during the summer of 1921 one of the authors could see a halo each morning, due to mucus having formed on the cornea. Emsley and Fincham (1922, p. 271).

(*d*) Leucocytes or fatty particles on the cornea are probably responsible for the rings that sometimes occur with conjunctivitis. Priestley Smith (1924, p. 153).

Here we have five substances suggested as the origin of the corona: blood corpuscles, spheres of pus, mucus, leucocytes, and fatty particles. I propose to review my observations to see if they throw any light on this problem.

On November 4, 1951, 3 days before I saw the corona which led to the long series of morning observations, I had considerable pain in my right eye due to the presence of a piece of grit which had to be removed by a doctor. For the next 20 days there was considerable irritation in the eye and during this period I saw a corona in one or both eyes on nine of the mornings (see Fig. 9). As I had only once before seen the corona, a year previously, I naturally associated its continued appearance with the wound in the eye caused by the grit. At this period I had not yet commenced to measure the size of the corona; but it was subsequently found that its size indicated that the diameter of the particles was 8μ. As this is the size of red blood corpuscles the association of the corona with the wound receives some support, although there was no visible sign of blood. The difficulty of this explanation is that the wound was in the right eye only, yet on four of the nine occasions

during this period on which a corona was seen in the right eye, it was seen equally bright in the left eye, in which there could have been no blood from the wound.

After the irritation had disappeared, there was a period of 40 days (Nov. 24–Dec. 3) in which, so far as I could tell, the eyes were perfectly normal. At first there were occasional appearances of the corona, on five mornings out of thirteen; then there was a period of eleven mornings without a corona, followed by eleven mornings only one of which was without a corona (in the latter period the coronas were more frequent and on the whole brighter than during the period after the grit had been removed); another period of six mornings without a corona brought the 40 days of normal eye conditions to an end. Here we see periods of bright coronas and of no coronas with nothing to give a clue to anything which might account for them.

On the morning of January 4, 1952, a stye was seen to be developing on the lower lid of the left eye. There was no corona on the morning of January 4 but poor coronas appeared in both eyes on January 5: the stye was not yet affecting the eye. Finding no corona on January 6, I examined the left eye and found a region of red discoloration below it. The stye had a yellow head, and after growing more and more irritable all day it broke in the evening and discharged a quantity of pus which was carefully removed with a swab. On waking on January 7, there was some resistance to raising the eyelids apparently due to moisture in both eyes. In a minute or two after starting the usual morning observation, coronas appeared in both eyes, that in the left eye somewhat the brighter. On January 8 the flow of pus from the stye ceased, but the left eye continued inflamed until January 11, during which period there was rather more moisture than usual in both eyes, but no deposit of solids in the corners. From January 7 to 11 there were bright coronas in both eyes. It is possible that pus could have got into the left eye during the 3 days, January 6, 7, and 8, when there was discharge from the stye, but it is difficult to see how it could have got into the right eye.

Before leaving the period affected by the stye, it should be pointed out that measurements of the coronas gave no further information about the nature of the particles. Good measurements of the size of the rings were made on 4 days between December 22 and 29, all more than 6 days before the stye appeared, and on 3 days, January 7, 8, and 9, during the discharge of the pus. The results were identical, showing particles of 8μ diameter: thus the size of the particles was the same in periods when there was not even the possibility of pus being present and periods when pus was present. This does not point to pus being the active agent in producing the corona.

The irritation due to the stye ceased on January 11 and afterwards to the end of the observations on March 28 there were mornings with coronas and mornings without; but no evidence was offered as to the possible origin of the particles.

The observations just described do not throw much light on the nature of the particles causing the corona. Blood possibly might have been responsible in the period after the wound, November 4–24, and pus in the period January 4–11; but then there would remain the difficulty that in each case coronas appeared in both eyes when only one could have been contaminated. This also leaves unexplained all those cases in which equally bright coronas were present in periods when the eye appeared to be normal and when there was certainly no discharge of blood or pus.

At one time I thought that the coronas were associated with the amount of moisture in the eye, but there were so many occasions with large amounts of liquid glare without coronas appearing that moisture cannot be the direct cause of the coronas. On the other hand normal moisture was associated with the periods following the wound and the stye; now these were periods of considerable direct irritation in one eye and sympathetic irritation in the other. Is it not possible that the coronas are associated with the irritation and not directly with the blood, pus or moisture ?

On January 21 I made the following note:

> These traces of coronas in the right eye may be associated with a slight prickling under the lid of this eye as though there were some grit between the lid and the eye. I only notice this when I wake up in the night.

During the remaining 3 months of the observations there are several references in my notes to this " prickling," and I summed up my thoughts on the matter as follows:

> The prickling in my right eye has continued since I first noticed it. It is clearly noticeable when I wake in the night and on first waking in the morning. I do not remember noticing it in the daytime. Once or twice I have felt a slight prick in the left eye; but in any case it is much less in the left than in the right. I have not been able to detect any correlation between the prickling and the appearance of the corona.

Combining these remarks on the prickling with those on the effect of irritation, I think a case can be made out for the prime cause of the corona being irritation in the eye, this would explain the coronas following the grit and the stye and also the greater frequency of coronas in the right eye than in the left eye. But this does not explain what the actual particles are and why they require several minutes to become active when the eyes open after several hours sleep. These are questions, however, for the ophthalmologist, rather than for the physicist.

Descartes' Description of a Corona

In his *Discours de la Méthode* (1637), Descartes described, with an illustration, a corona which he observed around the flame of a candle. The original was written in Latin; but I am translating from Victor Cousin's modern French version published in Paris in 1824.

> Finally, the cause of the rings which are sometimes seen around lamps and candles should not be sought in the air, but in the eye which sees them. Last summer I had

an experience which illustrated this very clearly. It was during a night voyage on a ship; I was making astronomical observations, watching the sky with one eye while keeping the other closed with the hand on which I was supporting my head. I had been observing all the evening when someone brought a lighted candle to the place where I was sitting.

On opening both eyes I saw two coronas (Lat. *coronantes*) around the flame [Fig. 10] of which the colours were as bright as I have ever seen in a rainbow; A B, the larger,

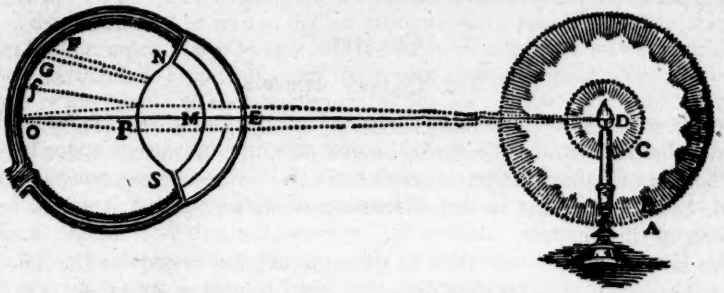

FIG. 10.—Descartes' diagram from "Oeuvres de Descartes" (1824), vol. 5, p. 292

was red towards A and blue towards B; C D, the smaller, was also red towards C, but towards D it was white and extended up to the flame. After that on closing the right eye I saw that the corona had disappeared; on the other hand on opening the right eye and closing the left they reappeared; this convinced me that the rings could be caused only by some disposition which my right eye had acquired while I had kept it closed.

There can be no doubt that Descartes had seen a corona similar to the corona we are discussing in this chapter. This comes out more clearly in the diagram than in the description. There can be no doubt to anyone who has seen the corona that the two shaded bands in Descartes' diagram are the two red bands. What Descartes calls the smaller corona, C D, is clearly the aureole with its red border. The description of his "larger corona" is not so satisfactory, for the expression "red towards A and blue towards B" is hard to interpret, and he gives no description of the colour of the clear ring between the two red rings; if we neglect "blue towards B" the rest of the description fits a corona consisting of a white aureole, and two red rings with a green ring between as shown in Fig. 8. It is very unlikely that Descartes made his drawing (or a sketch) while the aureole was still visible; it is much more likely he would draw it some time later from memory; if this is so it is surprising how nearly he has got the proportion between the radii of the rings correct as will be seen by comparing Descartes' corona with Fig. 8.

Descartes himself remarks:

> it is very common to those who have diseased eyes to see such coronas and they do not appear similar in all cases.

From this it is clear that coronas were well known in Descartes' time and probably they may have been mentioned in earlier writings, but there can be little doubt that Descartes was the first to give a description of any

entopical diffraction phenomenon accompanied by an attempt to explain it in what we today would consider a strictly scientific manner. So far as I know ophthalmologists have no definite name for the corona described in this chapter, and I have felt the need for one in writing the account of my observations. I therefore suggest that the name "Descartes Corona" could usefully be adopted as the scientific name for the corona which was first described by Descartes in 1637.

Chapter III

The Ciliary Corona

In my general description, in Chapter I, of the coloured rings which I see around lights I mentioned (p. 453) a disc of white light in the space between the source of light and the coloured rings. This I said was not part of the halo, and I postponed further discussion of it, saying that it would be the subject of this chapter.

We now know why the light in question cannot be part of the halo; for the halo is the first order spectrum of an optical grating, and there is no light in the space between the image of the source of light and the spectrum formed by a grating. When I came to describe to my friends the rings I see around lights and asked them to describe what they saw, I soon found that they also saw a glow around the same lights, but without any surrounding coloured rings. On making enquiries from ophthalmologists I learnt that this glow around bright lights is well known and is seen by all normal eyes; it is called the "ciliary corona." When I enquired the cause the only reply I could get was that it was due to "scattering," which is hardly a satisfactory answer. We have now to see if we can find the nature and cause of the ciliary corona.

Description of the Ciliary Corona.—In Fig. 2 (p. 454) I attempt to depict the ciliary corona as I see it within my lenticular halo. It consists of a disc of white light which is brightest near to the centre and decreases in brightness as the distance from the centre increases. On first sight this disc appears to have no particular texture, but on closer study it is seen to consist of a large number of fine white lines radiating from the central source of light superimposed on a background of innumerable points of light. These are so small that they are difficult to see individually, but produce the appearance of a uniform white surface. The word "point" is used advisedly here in order to reserve the word "spot" for use later when discussing much larger points of light. We must now study the corona in greater detail, discussing first the bright points of light and then the rays.

Points of Light in the Ciliary Corona.—When a white source of light is viewed by the naked eye, the points of light appear at one moment to be in rapid and agitated motion and then they suddenly stop and remain as though fixed in position. So long as the head is held still and the eyes focused on some feature in the field of view the bright points appear stationary; but if the head moves or the eyes wander the points set themselves into rapid motion. It is difficult to describe this motion, for although there is no recognizable pattern it is not entirely

irregular. The best description I can give is to liken it to a milling crowd of people, some moving in one direction and some in another; when viewed as a whole some portions of the crowd appear to have a general movement or surge in a definite direction, which direction is not long maintained, while other positions have surges in other directions. The bright points have a similar unco-ordinated motion; but occasionally the points in all parts of the corona move for a short time towards the centre and at other times away from the centre. Similarly radial motion can sometimes be seen in limited portions of the field. The only cases of regular motion which I have recognized take the form of circular waves originating at some particularly bright point on the source (probably points on the coiled filament of the lamp) and radiating outwards like the circular waves which spread over a sheet of water when a stick is moved backwards and forwards in the water near the bank. All these motions are most pronounced when one first looks at the light; for then it takes some time for the head and eyes to settle down. I have, however, failed to find any close relationship between any specified motion of head and eyes and the resulting movement of the points. When the points are in agitated motion the individual points appear to twinkle as well as to move; then, as the motion decreases, the twinkling also decreases, and when the motion has stopped entirely the brightness of the points remains constant.

When I examine the ciliary corona through my reading spectacles (11-inch focus, which ordinarily I do not wear when making these observations), the rays and the small points of light disappear, and are replaced by a small number of bright white spots. These spots have similar motions to those of the bright points, they are stationary when the head and eyes are fixed and are in rapid movement and twinkle when head or eyes are not at rest; their movement is much more striking because owing to their brightness it is more easily followed. I shall have to return later to these bright points and spots in an attempt to find their origin. In the meantime, the distinction between the " points " seen with the naked eye and the " spots " seen when spectacles are used, should be noted; the former are small and difficult to see, while the latter stand out like stars on a dark background.

Turning now to the rays seen in the ciliary corona, these are nothing like so prominent as those of which the lenticular halo is composed. As seen round the street lamp, they are not very conspicuous against the background formed by the bright points of light. It is seldom that a single ray can be traced from the centre to the edge of the corona; they have more the appearance of the individual hairs in a tress of hair, they lie in the same general direction, but are not straightened under tension. It is difficult to describe the rays in any detail, for the whole effect is near the limit of vision; but when examining the points of light and the rays intently I have the feeling that the rays are produced by the alignment of the bright points; and there is some colour effect as though the rays were built up of very short spectra; but the individual spectra cannot be studied for the whole effect is so elusive. As already stated, when the corona is viewed through spectacles the rays and points of light completely disappear, and are replaced by bright spots; it is as though all the light in the aureole, normally consisting of bright points and rays, is collected together into relatively few bright spots.

When sodium light was used at the National Physical Laboratory a yellow ring was seen with a radius of 3·1°, which is obviously the sodium line in the circular

spectrum forming the lenticular halo; we are not concerned with this ring at present as it is beyond the ciliary corona. Within the space surrounding the central source of yellow light, where the ciliary corona is normally situated, there were neither points nor rays as with white light, but a large number of bright spots of yellow light standing out sharply against a black background. The spots were most closely packed near the light, thinning out towards the edge of the disc. The spots were relatively large and bright, very similar to those seen with white light when spectacles are used and like them, sometimes stationary and sometimes in motion. When the intensity of the light was not great the whole cluster was well within the yellow ring of the halo. As the intensity of the source was increased the diameter of the cluster of spots became larger and reached the yellow ring of the halo, the position of which is of course not altered by the intensity of the light, and when the light had become very intense the boundary of the spots passed beyond the yellow ring and the brilliance of the whole phenomenon became too painful for prolonged viewing.

When mercury light was used there were two predominant rings in the halo, one green and the other blue, corresponding to the two main lines in the mercury spectrum. The phenomenon was similar to that for sodium light: there was a cluster of bright green and blue spots within the two rings of the halo, and the cluster expanded as the intensity of the source increased, finally passing beyond the two rings.

Exner (1877) showed that with white light the aureole of a corona due to uniform particles is a brilliant white disc, falling off in intensity from the centre and becoming red towards the border where its intensity becomes zero (see p. 465); if monochromatic light is substituted for the white light the whole aureole breaks up into a very large number of spots of monochromatic light which show up brilliantly against a black background.

This is exactly what occurs with the ciliary corona so that there can no longer be any doubt that the ciliary corona is the bright central portion (aureole) of a corona produced by particles of uniform size distributed at random somewhere in the substance of the eye.

A question which now arises is: why are there no coloured rings around the ciliary corona if it is the aureole of a corona ? The answer is clear: the intensity of the light in different parts of a corona due to particles is given in Table III, from which it will be seen that the intensity of the light in the first coloured band is only $1\frac{1}{2}$ per cent. of that near the source. As the ciliary corona, even near the source, is always faint compared with the intensity of the light in the source itself, the coloured band could not be seen unless the intensity of the source was so great that it would be intolerable to look at with the naked eye. This also accounts for another characteristic of the ciliary corona which at first sight seems strange. In all other coronas in which the coloured bands are seen, the size of the aureole and rings is independent of the intensity of the light; the size of the ciliary corona however expands and contracts as the intensity of the source increases and decreases. The explanation follows from what has just been said. As no light can ever be seen beyond the aureole, there must be a point on the radius of the aureole, where the light ceases to be visible, and this point moves outwards as the intensity of the light in source increases. Thus the apparent size of the ciliary corona is a function of the intensity of the source of light.

Location of Particles in the Eye.—Having now concluded that the ciliary corona is due to particles in the eye, we must consider where they are situated. Several ophthalmologists with whom I have discussed my observations have remarked that the wandering of the spots of light suggests the aqueous rather than the vitreous humour as the location of the particles. This is because they are under the impression that the spots are images of the particles; this, however, is a misapprehension, for a foreign particle in the eye cannot be "seen." The spots are certainly not images of particles moving freely about. In order to explain the formation of the spots of light in the aureole when monochromatic light is used, Exner suggested that, with a random distribution of diffracting particles, the light waves from the particles will not be evenly distributed over the retina, but will combine at certain places to produce amplitudes larger than average and so to give rise to the sensation of spots of light. Accepting this explanation and assuming no change in the position of the particles, there would be a change in the distribution of the light on the retina if there were any change in the path of the light waves within the eye. For example, any small muscular change affecting the crystalline lens would produce a redistribution of the light reaching the retina, involving a change in the position on the retina of the localities with large amplitudes. In this way an apparent movement of the visual spots would occur without any movement of the diffracting particles. This is not the place to develop this idea; but it illustrates the nature of the physical and physiological problems involved. With the amount of information available it is not possible to locate the position of the particles within the eye, and this question must remain over for further work.

Size of Particles causing Ciliary Corona.—If we could determine the radius of the ciliary corona we could calculate the size of the particles causing it. We have however seen that we cannot ever measure the radius of the aureole, as what is seen does not extend even so far as the limit of the aureole. We can, however, say that the true radius of the aureole is greater than the largest radius measured. In my experiments at the National Physical Laboratory the intensity of the sodium light source was increased to the limit of tolerance when the spots were already beyond 4° from the centre. This obviously gives a lower limit to the size of the aureole. Putting $n=1$, $\theta=4°$, and $\lambda=\cdot00059$ in Equation (2) (p. 466), we find the diameter of the particles, d, to be 0·01 mm. As the aureole was certainly larger than 4°, d must have been smaller than 0·01 mm., thus we finally arrive at this statement:

The diameter of the particles causing the ciliary corona cannot be larger than 0·01 mm. (10μ) although they may be much smaller.

Rays seen in Ciliary Corona

We must now turn our attention to the streaks or rays of white light which are seen in the ciliary corona. Similar rays are associated with other coronas produced by particles, and have been the subject of occasional studies by physicists. The most recent of these studies that I can find is that of de Haas (1918), who studied the development of the diffraction pattern as he increased the number of small holes, one by one, in a screen mounted to show the diffraction produced by the holes. His paper is not

entirely convincing, and the problem of the formation of the rays is still far from solved. As the observations I made on the rays of the ciliary and other coronas are I believe new and provide further information on the rays, I propose to describe them in some detail, so that they may be used by physicists in further work on this subject.

Radial Structure of Coronas produced by Particles.—In the following paragraphs I propose to discuss the rays seen in coronas produced by the random distribution of spherical or nearly spherical particles, chiefly the ciliary corona, but also those produced by lycopodium powder, blood smears, etc., the physical properties of which are known. Unless specially mentioned, the rays of which the lenticular halo is composed are not included, as these are produced by inequalities of the grating and are not directly diffraction effects.

Very soon after commencing my study of the ciliary corona, I noticed that the rays were much finer and brighter when the source of light was the filament of a clear electric bulb seen through a small hole, than when a pearl bulb was seen through the same hole. Some time later I noticed when using the pearl bulb that the rays became relatively brighter and more pronounced the further I removed away from the light. At first I did not connect these two observations. One day on observing the corona through a lycopodium disc, when I was using a pearl bulb behind a large hole as the source of light, I noticed that rays which were quite clear when the source was viewed from a distance of 5 or 6 ft. entirely disappeared when I approached to within 3 ft. of the source. At all distances nearer than this the corona consisted of aureole and coloured rings without any sign of rays either within the aureole or crossing the rings. A few rough tests showed that the distance from the source at which the rays disappeared as one approached the source was greater for large sources and smaller for small sources. Further and more accurate tests showed that, when a circular uniformly illuminated source of diameter d was viewed through a lycopodium disc from a distance l, the rays always appeared when d/l had the same value: in other words, when the diameter of the source subtended a certain angle at the eye, the rays appeared; with a larger angle there were no rays, with a smaller angle rays were always visible. I call this angle, φ, the "ray-formation angle," and the distance, l, between the eye and the source, the "ray-formation distance"; so that $d/l = \sin \varphi$.

With lycopodium, the ray-formation distance for a given source can be determined with little difficulty, for the change from rays to no rays is relatively sharp. Table VI contains the ray-formation angle for lycopodium

TABLE VI

RAY-FORMATION ANGLE FOR LYCOPODIUM

Diameter of source (mm.)	2	3	4	6	8	Mean
Ray-formation angle (min.)	23	24	22	23	23	23

powder for five uniformly illuminated circular sources of different sizes.

The next step was to make similar observations on the ciliary corona, for if the effect is the same it removes any lingering doubt as to whether the ciliary corona is a diffraction or a mere scattering effect. The first observation made with the naked eye showed at once that qualitatively the effect is the same; but it was not easy to get consistent numerical values. In the first place the rays are much less bright with the naked eye than with the lycopodium disc, and therefore it is much more difficult to fix the ray-formation distance. In the second place I found that looking at the bright light gave after-images which were troublesome. The measurements soon revealed a difference between the two eyes so that each eye had to be treated separately. After considerable experimental observation, a method was evolved which got over these difficulties and produced repeatable values.

The source of light was the surface of a 40-watt pearl electric bulb placed immediately behind a hole of the required size in a black screen. The number of sizes of the source to be examined was reduced to three, namely diameters of 2, 3, and 4 mm. The observations were made in a completely dark room. After fixing the source to be measured I waited in the dark until the eyes were dark-adapted. I then placed an eye-shade over the right eye and determined the ray-formation distance for the left eye; then I removed the eye-shade to the left eye and made an observation with the right eye. After waiting until any after-image had completely disappeared from both eyes, the observation was repeated with another size of source. I continued this procedure until I had made three observations with each eye on each of the three sources, this took about half an hour. If by this time the eyes were getting tired I took a few minutes rest before repeating the whole series. The complete series, consisting of six observations with each eye on each of three sizes of source, *i.e.*, 36 observations in all, took between 60 and 90 minutes. Table VII reproduces a set of observations

TABLE VII

RAYS IN CILIARY CORONA

Individual values of l and mean values of ϕ in one set of observations, December 28, 1951

Diameter of Source (d) (mm.)		2		3		4		Mean
Eye		Left	Right	Left	Right	Left	Right	
1	Individual Observations of l (cm.)	30	38	51	71	71	89	—
2		33	30	46	61	66	79	—
3		36	36	43	71	69	86	—
4		28	43	46	64	79	79	—
5		23	41	51	63	66	76	—
6		43	36	53	61	74	86	—
7	Mean l (cm.) ...	32	37	48	65	71	83	—
8	Sin $\phi = d/l$ ...	·0062	·0054	·0063	·0046	·0056	·0048	—
9	Mean ø (min.) ...	21	19	22	16	19	17	—
10	Ratio ϕ_L/ϕ_R ...	1·11		1·38		1·12		120
11	Mean $\frac{1}{2}(\phi_L+\phi_R)$ (min.) ...	20		19		18		·19

made in this way, in which separate columns are given for the left and right eye separately; the order of the observations was as shown in the Table, starting at the left-hand end of the first line and ending at the right-hand end of line 6. The whole set of 36 observations in this case took 65 minutes.

Lines 1–6 give the individual values of the ray-formation distance (l) for each eye, and three sources of different sizes. Considering the difficulty of the observations, the individual numbers in the separate columns do not show an unduly large scatter. Line 9 shows that the mean values of the ray-formation angles for the two eyes are somewhat different; but line 10 shows that the ratio between the eyes is practically constant. Line 11 shows that the ray formation angle, using the mean for the two eyes, is practically independent of the size of the source. This however is only the result from one day's observation. Observations by this method were repeated on three successive days and are summarized in Table VIII; they proved to be so consistent that I did not feel it necessary to take further observations.

TABLE VIII

RAYS IN CILIARY CORONA

Mean Values of Ray-formation Angle, ϕ

Diameter of Source (mm.)	2		3		4		Mean	
Eye	Left	Right	Left	Right	Left	Right	Left	Right
Mean ϕ (min.)	21	18	21	16	19	17	20	17
Ratio ϕ_L/ϕ_R	1·17		1·31		1·12		1·20	
Mean ϕ (min.)	20		19		18		19	

From this Table we see that for the ciliary corona in my eyes the angle subtended by the diameter of the source at the eye must be smaller than 19′ for rays to be seen; some slight difference is shown in the two eyes—left eye 20′, right 17′—this difference is real; but its significance will remain unknown until we know more about the origin and physics of the rays.

I have not had the opportunity of testing other eyes in the same complete manner; but a few tests on three of my friends, both of lycopodium and ciliary coronas, showed that they had no difficulty in determining the ray-formation distance and their measurements were numerically comparable with mine.

In addition to the lycopodium and ciliary coronas I made similar measurements on the Descartes corona (seen on waking) and on that produced by a smear of blood on a glass slip. The results for these four coronas are collected in Table IX (opposite). The diameters of the particles causing the coronas are given in the last column.

It will be seen that the ray-formation angle, φ, is in each case found to be independent of the diameter of the source; there is, however, a considerable variation for the different coronas in the size of this angle, from 19′ for the ciliary corona to 37′ for the blood corona. One's first idea is that φ would

TABLE IX
RAYS IN FOUR DIFFERENT TYPES OF CORONA
Value of ϕ

Diameter of Source (mm.) ...		2	3	4	Mean	Diameter of Particles
Type of Corona	Lycopodium ...	23′	24′	22′	23′	30μ
	Descartes	29′	28′	26′	28′	9μ
	Blood smear ...	43′	35′	32′	37′	7μ
	Ciliary	20′	19′	18′	19′	<10μ

depend in some way on the size of the particles; but the diameter of the particles forming the corona (last column), appear to have no relationship with the mean values of φ in the previous column. Considerable light is thrown on this aspect by the observations made on the Descartes corona. On six mornings I was able to measure the ray-formation angle for this corona as well as the radii of the rings, thus obtaining simultaneous values of the ray-formation angle and the size of the particles (Table X).

TABLE X
RAYS IN DESCARTES' CORONAS
Mean values for ray-formation angle, ϕ, on six mornings

Date	No. of Measurements	Diameter of Source (mm.)				Diameter of Particles
		2	3	4	Mean	
December 23 ...	4	35′	35′	34′	35′	9μ
December 26 ...	2	17′	19′	18′	18′	9μ
December 29 ...	2	27′	20′	21′	23′	9μ
January 7	3	27′	27′	25′	26′	9μ
January 10	3	38′	41′	36′	38′	9μ
January 12	3	30′	24′	22′	25′	9μ
Mean		29′	28′	26′	28′	9μ

The first thing to notice in Table X is that the ray-formation angle varied largely from day to day (column 6); that these variations are real is shown by the fact that the same angle was found for all three sources on any one morning (columns 3, 4 and 5). If these variations from day to day had been due to variations in the size of the particles this would have been shown by variations in the radii of the rings (column 7), which depend on the size of the particles. But in all six cases no measurable difference was found, showing that the size of the particles remained constant. Thus we can definitely say that the size of the ray-formation angle is not determined by the size of the particles causing the corona.

All the rays we have discussed so far are associated with coronas produced by particles distributed at random between the source of light and the retina. Rays are however a feature of the lenticular halo, and these also disappear if one approaches close enough to the source. The ray-formation angle, φ, for the halo is 39′, which is to be compared with the angle for the coronas

given in Table IX. It is larger than the angle for any of the coronas, but only slightly larger than that for the blood corona, namely 37′. There can be little doubt that de Haas is right in attributing the rays in the coronas to secondary diffraction effects between the diffracting particles; there can be still less doubt that the rays in the halo are due to the irregular distribution of the fibres which form the lenticular grating. Why the rays produced by these two different physical effects should both have a constant ray-formation angle, is another problem for physical research.

Origin of Bright Streamers seen radiating from Intense Sources of White Light.—If one looks at the reflection of the sun in a sheet of glass or in the surface of a puddle it can be viewed for a short time without discomfort. The sun will then be seen as a clear cut disc of light with a perfectly sharp edge, there will be no rays radiating out from it. The same is true of the moon, the disc is clear without rays of any kind. We now know the reason for this, the angle subtended at the eye by both sun and moon is 30′ while the ray-formation angle for the ciliary corona is 19′, thus both sun and moon are too large when seen from the earth to produce rays. If now we examine the image of the sun in a convex or concave mirror, the bowl of a silver spoon will do very well, we see it surrounded by innumerable brilliant rays streaming out to a distance of several degrees from the centre. The mirror has reduced the apparent diameter of the sun to such an extent that the angle it subtends at the eye is well below 19′, and the ciliary corona now consists almost entirely of rays. That it is the reduction in the apparent size of the sun, and not the reflection from the mirror, which has induced the ray-formation, can be seen by breaking the original sheet of glass into small pieces. Each piece is now too small to reflect the whole disc of the sun, but can only reflect a portion, and if this portion subtends at the eye an angle of less than 19′, the rays will be seen. The same effect is seen when one catches glimpses of the sun between the leaves as one walks in a wood. The portions of the sun glimpsed in this way are accompanied by numerous rays. In this case no reflection is involved.

At the National Physical Laboratory I was shown an intense source of electric light the area of which had been reduced to a mere pin-point. It was the centre of a most brilliant disc composed entirely of the finest streamers of white light.

The rays or streamers seen round brilliant small sources of light are therefore a diffraction effect, produced by the same particles within the eye as produce the glow seen round every bright source of light (the ciliary corona); they are not shown when the point of light is photographed in a camera.

Summary

The author describes in detail three ocular diffraction phenomena and discusses the physics of their formation based on observations made on his own eyes.

(*a*) It is confirmed that the coloured rings sometimes called the lenticular halo form a circular spectrum produced by the radial fibres of the crystalline lens, which become sufficiently uniform in size and regular in arrangement near to the periphery of the lens to act as an optical grating. The angular

radius of the yellow ring ($\lambda = 0.00057$ mm.) is found to be 3.1° and the radii of the other coloured rings to be in linear proportion to their wavelength. The optical measurements give 8 μ as the width of the fibres which are optically active to within 1 or 1.5 mm. of the centre of the lens. Methods of differentiating the lenticular halo from other coloured rings are discussed.

(*b*) Observations of the corona first described by Descartes in 1637 and usually seen within a few minutes of waking were made each morning during 5 months. This corona consists of a bright white central aureole surrounded by three broad coloured rings: the first red, the second green, and the third red; traces of further red and green rings could sometimes be seen. The central radii of the first three rings are 4.1°, 6.0°, and 8.6° respectively; from this it is deduced that the corona is caused by particles with diameter of 8 μ. Experiments show that these particles are present on the anterior surface of the cornea; they cannot be removed by blinking or rubbing but are immediately dissolved in a bath of distilled water or saline solution. The nature of the particles is discussed without any conclusion being reached. The name "Descartes corona" is suggested for these rings.

(*c*) The glow seen around bright lights is called the ciliary corona because it is frequently seen to be composed of bright fibre-like rays of light. It is shown that this corona is also a diffraction effect, the rays becoming visible when the source of light subtends an angle of at least 19′, *i.e.*, when the distance of the light from the observer is greater than 180 times its diameter. The diffraction is due to particles of uniform size (diameter less than 10 μ) somewhere in the body of the eye. The nature of these particles and their exact location in the eye are as yet undetermined.

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APPENDIX

METHOD OF MEASURING THE COLOURED RINGS

The following has been found a convenient method of measuring the radii of the coloured rings.

A wooden box of 18″×12″×6″ is used; the vertical front 18″×12″ is made of thin brass sheet, hinged at the top with the edges turned over the sides and bottom to form

a light-tight door to the box. In the centre of the brass plate is a circular hole ($\frac{1}{8}$" in diameter) and in the horizontal line through this hole there are sixteen holes ($\frac{1}{10}$" in diameter) spaced at intervals of an inch, eight on each side of the central hole and numbered 1 to 8 from the centre outwards; the fifth hole on each side has two additional holes, one above and one below, to facilitate counting the holes in the dark. There is a similar series of small holes on the vertical line through the central hole extending five inches above and below the central hole; Fig. 11 shows the arrangement of the holes. The brass plate forming the front of the box is painted black.

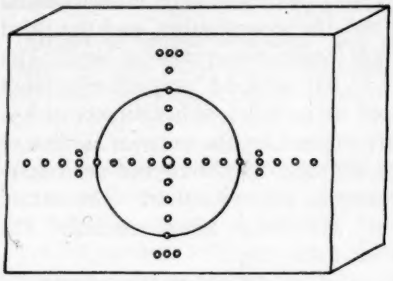

FIG. 11.—Box for measuring rings.

Behind the central hole, within the box and nearly touching the front plate, is a 100-watt pearl electric bulb which is mounted on a brass rod screwed into the bottom of the box so that the lamp may be raised and lowered to bring the brightest part immediately behind the hole. Thus the central hole is brilliantly illuminated and forms the "bright source of light" under observation. A sufficient thickness of thin card is pasted on the back of the brass plate within the box over the smaller holes to cut down the light from the interior of the box to the minimum necessary for the small holes to be just visible in the dark.

Between the central hole and each of the small holes to the left and right short pieces of wire are soldered and bent to form hooks to either of which the end of an inch tape-measure can be attached.

Before making a measurement the height of the box is adjusted so that the central spot is at the level of the observer's eye, and the tape-measure is attached to the hook on the same side of the central spot as the eye to be tested—say the left-hand side. When the observer stands in front of the box he sees the central bright spot surrounded by the coloured rings which contract and expand as he moves towards or away from the box. He takes the tape measure in his left hand and approaches the box until the ring he is measuring is seen between the central spot and the first small spot. He then moves backwards until the ring coincides with spot No. 1 on one of the rows of spots and checks this by seeing that the corresponding spots on the other three rows are also on the ring. He then raises the tape-measure, holding it lightly between thumb and first finger some distance short of the eye, and brings it into contact with the corner of the eye by sliding the thumb and finger along the tape. The room light is then turned up and the position of the grip on the tape-measure read off and recorded.

The observation is then continued, using other spots in turn, until sufficient measurements have been made to give the accuracy required.

If it is required to make observations with different sized sources it is easy to have the central hole enlarged and a frame soldered around it into which brass slides, each with a different sized hole can be slipped.

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DEGENERATIONS OF THE DOG RETINA*

II. GENERALIZED PROGRESSIVE ATROPHY OF HEREDITARY ORIGIN

BY

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A SPONTANEOUS degeneration of the retina of the dog, due to a generalized progressive atrophy of the neuroepithelium and called " night blindness " or retinitis pigmentosa of dogs has been known for nearly half a century, since Magnusson's recognition of the syndrome in Gordon Setters in Sweden about 1905 (Magnusson, 1909, 1911, 1917); he considered the condition to be similar to retinitis pigmentosa in man. More recently a somewhat similar syndrome has been reported in Red Irish Setters in England (Hodgman and others, 1949; Parry, 1951). These workers described in detail the clinical stages of the syndrome and presented evidence indicating that the disease was inherited as if determined principally by a recessive autosomal Mendelian factor.

The present paper presents data obtained in a series of matings under controlled conditions designed to establish whether or not the syndrome is hereditary, to provide material for detailed histological and electrophysiological studies, and to establish the relationship of the canine syndrome to human retinitis pigmentosa.

Materials and Methods

The retina of the normal dog, the details of the management of the dogs, and the methods and terminology used, were described in the first paper of this series (Parry, 1953a).

The dogs were pedigree Red Irish Setters. About 120 dogs were used, including 93 in sixteen litters born and reared in isolated premises at the Research Station, of which 58 were affected with the degeneration and 35 unaffected.

The condition of the eyes was established by clinical ophthalmoscopy, vision tests, and pathological examinations of animals at varying stages of the disease. The objective test for the disease described by Parry, Tansley, and Thomson (1951; 1953), namely inability to detect the electroretinogram, was used on thirty dogs.

All the matings were carried out in our isolation kennels by an experienced kennelman to reduce to a minimum the chance of promiscuity.

All the animals were known to be " affected " (with defects of vision and carrying the recessive gene in a homozygous state), " carriers " (with normal vision but carrying the recessive gene in a heterozygous form), or " clear " (with normal vision and not carrying the recessive gene), according to the criteria previously given (Parry, 1951). By mating " carrier " and " affected " parents, mixed litters of affected and unaffected pups were produced, the unaffected pups serving as controls for management and other factors which might give rise to acquired forms of retinal degeneration.

Special care was taken to exclude other forms of blindness known to occur in dogs, so far as present knowledge permits. Apart from the retinal defect and the absence of an electroretinogram, the criteria of normality were similar to those described previously

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(Parry, 1953a). In addition, disease of the optic pathways and visual cortex was excluded by clinical and pathological examinations, and particular care was taken to ensure that the other types of retinal degeneration (to be described in later papers of this series, Parry, 1953b etc.), some of which have been found in Red Irish Setters, were not present. All the dogs mated in a " test-mating " (Hodgman and others, 1949) produced affected and unaffected offspring in the numbers one would expect if they were suffering from the simple hereditary disease only.

Results

(1) Changes in the Eye

The disease can be considered conveniently in the three stages (Hodgman and others, 1949) in which vision progressively disappeared. During Stage 1 night vision was gradually lost but day vision remained normal. During Stage 2 the dog was completely blind at night and gradually lost its powers of day vision. In Stage 3 the dog was completely day and night blind and cataract often developed. All dogs showed a similar stage of the disease in both eyes and all parts of the fundus were affected to a similar extent. The progress of the disease is summarized in Table I.

TABLE I

SIGNS AND LESIONS OF THE THREE STAGES OF THE HEREDITARY

Stage		Age	Vision Defects		Ophthalmoscopy	Pupil	
			Night	Day		Average Size (mm.)	Light Reflexes
1	*a*	3–6 wks	?	0/5	Media not yet clear	4/10	++
	b	2–6 mths	2–4/5	0/5	Swirling of supra-tapetal retina, tapetum shows increased reflectivity and crystalline texture	5–7/12	++
2	*a*	3–18 mths	5/5	1–2/5	As Stage 1*b* with loss of secondary retinal blood vessels Optic papillae still normal Non-tapetal fundus pavement grey	6–8/14	++
	b	5–24 mths	5/5	3–4/5	Changes more marked and optic papilla pale owing to loss of secondary arterioles	8–12/14	+
3	*a*	6 mths to 3 yrs	5/5	5/5	Only three main blood vessel groups and chief collaterals remain Tapetal reflectivity greatly increased	12/14	0
	b	1–4 yrs	5/5	5/5	Little change Pigment of non-tapetal fundus may become patchy	12/14	0

Stage 1.—This stage falls into two parts:

(*a*) before the development of normal powers of vision at about 6 weeks of age (105 days post-conception) when the retinal degeneration can only be detected by electroretinography or histology,

(*b*) after the age of 6 weeks when clinical methods can also be employed.

Stage 1*a*.—The post-natal differentiation of the retina proceeded as in normal eyes. Eyes of known affected pups could not be recognized as abnormal earlier than about the 78–80th day post-conception (15th to 20th day after birth), when a small number of pyknotic nuclei, with rounded densely-staining chromatin, appeared in the outer nuclear layer, five to ten in each field of about 5,000 nuclei (incidence of 0.1–0.2 per cent.). These pyknotic nuclei showed up well in Feulgen and azan stained preparations. By the 80–85th day post-conception the outer limbs failed to develop their regular palisade arrangement, and the number of pyknotic nuclei rose to 20–25 per field (0.4–0.5 per cent.). These pyknotic nuclei disintegrated completely and were resorbed without the appearance of inflammatory or phagocytic cells. At the same time the contiguous nuclei moved together and took up the space previously occupied by the lysed nucleus, thereby causing a gradual reduction in the width of the outer nuclear layer and the number of nuclei in each radial palisade. By about the 90th day post-conception (4 weeks after birth), the outer

GENERALIZED PROGRESSIVE RETINAL ATROPHY IN RED IRISH SETTERS

Electro-retinogram	Histology	Figure	Approx. Widths of Retina in μ, area centralis 5 mm. from Optic Papilla		
			Total	ONL	RCL
Absent after 30 days old	A few pyknotic rod nuclei in the outer nuclear layer with some loss of substance in the outer limbs	—	200–220	50	12–15
Absent	Reduction of outer nuclear layer to 2–3 nuclei, loss of all outer limbs and of rod inner limbs, those of the cones being shortened	—	120–130	17–19	12–15
Absent	No rods or rod nuclei remain; outer nuclear layer reduced to cone nuclei 1–2 thick; inner nuclear layer and ganglion cells unaffected; cone inner limbs gradually lost	1	100–120	12–15	5–6
Absent	A few cone inner limb segments remain but these disappear and the external limiting membrane becomes adjacent to the pigment epithelium	2	120–130	10–12	3–4
Absent	Layer organization of retina retained in parts, where loss of inner limbs retarded; inner limbs absent in foci, but cone nuclei remain as distinct layer; inner nuclear layer little affected, apart from some increase of cytoplasmic bodies of Müller's cells; ganglion cells still normal	3, 4	80–120	7–8	—
Absent	Loss of layer organization; marked reduction in width of retina; retina sclerosed with gliosis and remnants of nuclear layers; very occasional pigment cells; loss of pigment epithelium	5	30–80	—	—

nuclear layer was reduced to seven to eight nuclei, the outer limbs were more fragile than usual, and the electroretinogram was not detected. Thus by the 6th week of life, when opthalmoscopic and visual tests could be applied, the atrophy of the rods and their nuclei was already advanced.

Stage 1b.—Progressive loss of night vision occurred. At 6 weeks of age, on a night with a moon or much star-shine, affected dogs could still find their way about, but when 3 to 4 months old they lost all powers of night vision even at dusk. Day vision, the eye preservation, and pupillary light reflexes were normal, but the average pupil size increased slightly from 5 to 7/12 mm. as night vision deteriorated.

In the fundus there was some loss of the granular " beading " of the tapetum, the reflection from which was increased, usually with an augmented green component in the colour of the reflection. The supra-tapetal tissues showed a disturbance of refraction called " swirling ", *i.e.* on examining the fundus by direct retinoscopy, the supra-tapetal tissues were fully transparent but there was a ripple-like distortion of the tapetum not unlike that produced on the surface of a quiet pond by the dropping of a small pebble.

In histological preparations the retina was markedly reduced in width, chiefly because of the progressive loss of rod nuclei. At the end of this stage the outer nuclear layer consisted of a layer of cone nuclei two to three deep, with an occasional rod nucleus. Coinciding with the reduction of the outer nuclear layer, the rods and cones were progressively reduced in width, the loss of the outer limbs being followed by that of the inner limbs of the rods, until only the inner limbs of the cones foreshortened to 1/3 of their normal size remained.

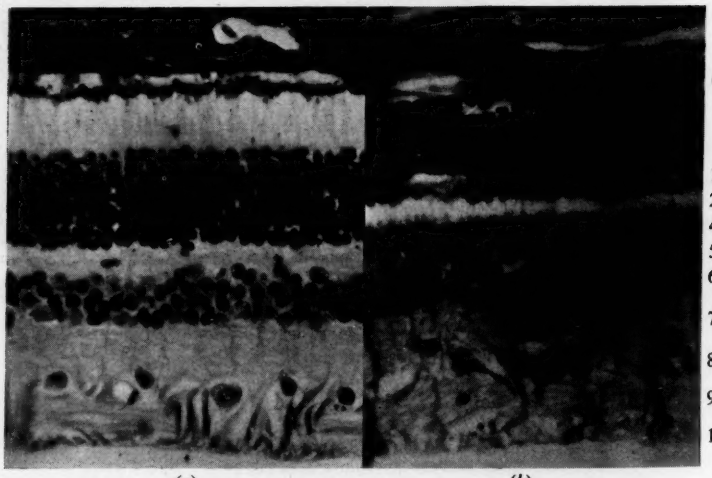

FIG. 1.—Sections through retinae of two 3½-months-old dogs, litter-mates: (*a*) normal, (*b*) showing Stage 2*a* of the atrophy; non-tapetal fundus. Note the great reduction of the outer nuclear and bacillary layers in the affected pup without changes in the inner layers of the retina. There is almost complete loss of rod nuclei, leaving only cone nuclei in the outer nuclear layer. The bacillary layer is reduced to the ragged remnants of a few cone inner limbs. Intra-vital fixation with Kolmer's cold-blooded fluid. Heidenhain's iron haematoxylin (x 270).

Stage 2.—The dogs were completely night blind, but they retained some powers of day vision for periods of from 3 to 18 months, during which time the average pupil size increased gradually from 6 to 10/14 mm., but the pupillary light reactions though sluggish were not lost until day vision was also lost. There was a progressive loss of the stellulae of Winslow, the swirling of the supra-tapetal tissues became more marked, and the texture of the fundus lost its granular beading and became crystalline, *i.e.* it appeared to be composed of large rectangular crystals.

There was some general loss of secondary blood vessels of the fundus. At the same time the non-tapetal fundus became greyer and its blood vessels lost their "silver-wiring" reflection. The pupilla also became much paler and the secondary blood vessels disappeared. During the early part (Stage 2*a*) the changes of fundal texture occurred with only slight loss of blood vessels or deterioration of the pupil's response to light, but during the later part (Stage 2*b*) the loss of blood vessels was marked and the response of the pupil to light deteriorated rapidly.

Histologically, Stage 2 was characterized by the final disintegration of the cones, although the cone nuclei and the general organization of the retina remained. During Stage 2*a*, the stubby remnants of the cones (Fig. 1) gradually disappeared until in Stage 2*b* the external limiting membrane came to lie against the pigment

FIG. 2.—Section through retina of 6-months-old dog affected with Stage 2*b* of the atrophy; periphery of tapetal fundus. Note the complete loss of rod nuclei. The remnants of the cones form a thin layer between the atrophic pigment epithelium and the thickened external limiting membrane. The inner layers of the retina still remain normal, but the nuclei of Müller's cells stain more deeply. Note the trans-tapetal capillary, chorio-capillaris, and retinal blood vessels, which remain normal. (This dog still had pupillary light reactions and some day vision, defect about 3/5.) Intra-vital fixation with Kolmer's cold-blooded fluid. Heidenhain's iron haematoxylin (x 450).

epithelium, the cells of which were flattened and losing their pigment (Fig. 2). The outer nuclear layer consisted of cone nuclei only and the outer fibre layer was much thinner. Müller's cells were more conspicuous and their nuclei stained more deeply, but the inner nuclear layer and the layers within were otherwise apparently normal. It appeared that the loss of the rods from the bacillary layer caused a gradual disintegration of the cones without loss of their nuclei. The density of the cones and of their nuclei varied slightly in different dogs but was remarkably regular in any one retina.

Stage 3.—This stage began with the complete loss of day vision and the pupillary light and eye preservation reflexes. Dogs usually showed considerable apprehension when this occurred but most of them adjusted themselves to the loss of visual perception within a week or so. An occasional dog remained very apprehensive and timid indefinitely, but most dogs moved about freely in their usual surroundings and were not molested by their normal kennel companions.

For the first few months (Stage 3*a*) the fundus remained little changed except that only the main blood vessels and their larger collaterals were visible. However, the previous location of the branches could be seen as white " ghosts ", particularly in the non-tapetal fundus. The papilla was almost white, its blood vessels reduced to the three main arteries and veins, and its margin ragged and indistinct; its surface flat, level with the surrounding fundus and without any cupping. The reflectivity of the tapetum was very marked and associated with a very striking crystalline texture, but the fundus showed a distortion of the ophthalmoscope light at the periphery of the beam where the normally transparent vitreous chamber was now a cloudy white, presumably because of scattering of the light rays by the vitreous and/or at the surface of the retina. The non-tapetal fundus showed a deeper grey appearance, but in portions of the more ventral quadrants this was missing and areas of darker brown pigmentation were visible, although these appeared to be no more than the normal choroidal pigment free of the grey " overwash ".

During the later stages (3*b*), the examination of the fundus was usually hampered by the development of cataracts, but in three animals affected with this stage for 2 years or more spontaneous luxation of one or both lenses occurred and thus permitted examination of the fundus. In these animals the blood vessels had almost disappeared, only short portions of the main groups being visible. The disc was very ragged and of a grey-white colour. The tapetum was less highly reflective, but remained a continuous sheet without signs of unusual pigmentation. The non-tapetal fundus showed greater unevenness of pigmentation and the darker areas were more numerous. In one dog choroidal pigmentation was so markedly reduced that the choroidal blood vessels could be seen lying in the middle of five or six light beige-coloured streaks about 8 mm. wide (after magnification) radiating fanwise from the papilla from which the normal brown pigment was completely missing. These streaks had not been visible in this dog 3 years previously before the cataracts developed.

For the first few months after complete loss of day vision (Stage 3*a*) the layers of the retina could still be distinguished in sections, although the outer nuclear layer was reduced to an interrupted layer of cone nuclei, one to two thick; these were round but their chromatin was lobulated more like that of the rod nuclei; they still retained a positive Feulgen reaction and stained bright red with the azan

FIG. 3.—Section through retina of 4-year-old dog, which had gone completely day-blind 4 months previously, showing Stage 3*a* of the atrophy; non-tapetal fundus. The normal layers are still present on the left, but the pigment epithelium, bacillary, and outer nuclear layers have disappeared on the right. The nuclei of the inner nuclear layer show pyknotic change and their cytoplasmic processes are more conspicuous. The ganglion cells are losing their Nissl's substance. The fibre layers are but little affected. Intra-vital fixation with Kolmer's cold-blooded fluid. Heidenhain's iron haematoxylin (x 350).

method, and this served to distinguish them from nuclei of the inner nuclear layer which still appeared normal (Fig. 3). The rods and cones had disappeared, although occasionally there remained small pockets up to 50μ long of the inner limb segments of the cones. The inner nuclear layer was almost normal, except that the fibres of Müller's cells were more prominent and their nuclei stained very darkly with basic dyes. The ganglion cells were remarkably little affected, although their Nissl's granules appeared to be disintegrating slightly. The pigment epithelium cells were little changed in shape, although they were entirely devoid of pigment, even over the non-tapetal fundus; no pigment accumulations were noted in the retina itself. The chorio-capillaris was very much reduced, many of the tapetal cells had lost their nuclei and their cell outline, and the trans-tapetal capillaries were reduced in number. Many retinal blood vessels were still present but more deeply buried in the substance of the retina; occasionally capillaries passed through the outer nuclear layer from the retinal circulation to join the choroidal (Fig. 4, overleaf). In Stage 3*b* these changes proceeded to definite sclerosis; the remaining cone nuclei were interspersed with the remnants of the outer nuclear layer to form an irregular single nuclear layer about three to four

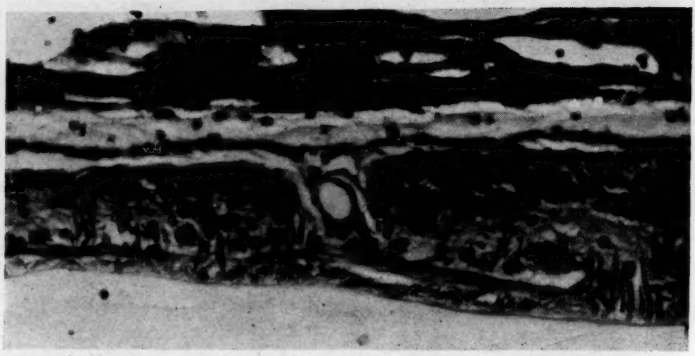

FIG. 4.—Another part of same section as that seen in Fig. 3, showing a focus of more advanced degeneration with loss of layer organization; tapetal fundus. The nuclear layers are disintegrated, but remnants of the pigment epithelium and optic nerve fibres remain. Note the blood vessel joining the retinal and choroidal circulations (x 240).

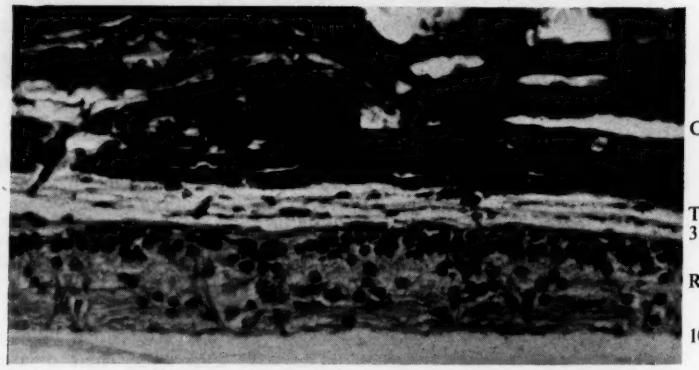

FIG. 5.—Section through retina of 6-year-old dog which had been completely blind for 4 years, showing Stage 3*b* of the hereditary atrophy; non-tapetal fundus. The layer organization is completely lost, most of the nuclei have disappeared, and the pigment epithelium is missing, so that the remains of the retina are in direct contact with the tapetum. Note the absence of pigment cells in the sclerosed retina. Intra-vital fixation with Kolmer's cold-blooded fluid. Heidenhain's iron haematoxylin (x 240).

nuclei thick in a matrix of glial fibres, the whole retina being reduced to 15–20μ thick (Fig. 5). The cone nuclei stained brown with azan like those of the inner nuclear layer, except that their nucleoli still stained bright red. The walls of the retinal arterioles were much thickened, but the larger vessels were still present in their usual frequency, although buried in the substance of the retina. This would account for the apparent loss of the secondary retinal vessels on ophthalmoscopic examination. In places, the nuclear layers were interrupted by glial fibres running from the inner to the outer surface of the retina. Very occasionally an odd round pigmented cell was found, but these were usually single

and not disposed near any blood vessels. As the pigment epithelial layer had atrophied completely, these pigmented cells had probably arisen from choroidal pigment cells which had lost their elongated form and become round. The pigment in the choroid became aggregated into larger masses and the cells became more rounded. In cases totally blind for 2 to 3 years there was definite thinning of the choroid to half its normal thickness or less with reduction of its blood vessels, but the sclera remained little affected.

(2) Rate of Development of the Disease

The earliest signs were always detectable by ophthalmoscopic examination and by night vision tests when the dog was 6–8 weeks old. Thereafter the rate at which the fundus changes and the visual defects progressed varied somewhat. In one family strain, all affected dogs lost all powers of night vision by 3 months of age and were completely day blind by 6–8 months. In another family, night vision was not lost until 6–9 months of age and some day vision remained until they were over 3 years old. When an animal of the first strain was mated to one from the second, the syndrome tended to develop rapidly in their offspring. We have not seen any case of spontaneous arrest or remission of the disease.

(3) Associated Defects

The outstanding defect associated with the retinal disorder was cataract. During the late stages of the disease the majority of affected dogs over about 2 years of age developed complete bilateral nuclear cataracts. Small subcapsular cataritic foci 1–2 mm. in diameter were first detected in some dogs at 8–9 months old; these foci enlarged and gradually became denser until the lens was completely opaque. Of eight animals which lived more than 2 years, seven developed bilateral cataracts. In the older animals these tended to become luxated, particularly in active dogs which were not afraid to move about and were hence more liable to damage their eyes.

One dog only showed bilateral myopia, a +8 D. lens in the ophthalmoscope being necessary to bring the fundus into focus.

The oestrous cycle and breeding performance of affected bitches showed no disturbances which could in any way be attributed to the retinal degeneration. Five bitches totally blind for 2 to 4 years continued to come into season and breed regularly. Male dogs also maintained their breeding performance, one during 4 years of complete blindness; however, one which became extremely apprehensive with the onset of blindness and did not adjust itself to the complete loss of vision, became of little use as a stud dog owing to this nervousness, although a litter brother was used at stud for 2 years after complete loss of vision.

The growth and general behaviour of the affected animals were normal; some would have been potential show winners. No other neurological defects were noted, but some dogs were probably of subnormal general intelligence; some strains of Red Irish Setters known to carry the gene appeared to be less intelligent than strains not carrying the gene, and these strains were less readily trained as gun-dogs.

Should any other defects of the nervous system be closely associated with the gene, they would have been likely to appear in these dogs as 4 of the 16 litters were the result of litter brother-litter sister matings.

(4) INHERITANCE OF THE SYNDROME

Four affected dogs, four affected bitches, three " carrier " bitches, and three " carrier " dogs, known to be homozygous and heterozygous respectively with regard to the gene, and one " clear " dog were used for the matings reported in this paper.

Sixteen litters were born and 58 pups affected with the syndrome were reared to an age when a certain diagnosis could be made on the basis of clinical, microscopic and/or electroretinographic data; in nearly every case the clinical and electroretinographic diagnosis was supplemented by histological study.

The results of these controlled matings are shown in Table II. It will be seen that the mating of two affected parents always produced all affected offspring with approximately equal numbers of males and females. The mating of a " carrier " with an affected animal produced mixed litters of affected and unaffected animals in approximately equal numbers and equally divided between the sexes. The mating of an animal not carrying the gene with an affected animal produced no affected offspring.

TABLE II

INCIDENCE OF AFFECTED AND UNAFFECTED PUPS BORN OF DOGS CARRYING *THE GENE FOR HEREDITARY RETINAL ATROPHY*

Type of Mating		*Litters*	Offspring						Ratio of Affected to Unaffected
			Affected			Unaffected			
Male	Female		Total	Male	Female	Total	Male	Female	
Affected	Affected	6	36	16	20	0	0	0	36 : 0
Affected	*Carrier*	5	10	9	1	23	13	10	22 : 29
Carrier	Affected	4	12	3	9	6	2	4	
Clear	Affected	1	0	0	0	6	1	5	0 : 6
Totals		16	58	28	30	35	16	19	

These data confirm the view based on field observations and expressed previously (Hodgman and others, *1949) that the syndrome is a hereditary* one and that it is inherited principally as an autosomal recessive Mendelian factor. The data provide no grounds for assuming any coupling or repulsion *of the factor* to either sex chromosome, nor of modifying genes of any practical importance.

In the fifty dogs which came to autopsy, the optic nerves and brain were examined carefully for signs of macroscopic abnormalities. None was *found. Detailed studies of the centripetal degeneration which* might be expected to follow loss of the retinal ganglion cells in Stage 3 of the disease were not attempted.

Discussion

The retinal degeneration observed in these Red Irish Setters is very similar to that described by Bourne, Campbell, and Tansley (1938) in the rat, and by Tansley (1951) in the mouse, the chief difference being the more rapid progress of the disease in those species. Study of the degeneration in the dog has, however, enabled us to extend our fundamental knowledge of the disorder by correlating the results of retinoscopic examinations and vision tests with histological data, and by applying an objective measure of function in the form of the electroretinogram, techniques which are difficult to apply in the young rat and mouse.

The use of these techniques and the rather slower rate of development of the degeneration in the dog has allowed us to establish that the retina does become functionally mature, as judged by the electrophysiological evidence of an immature electroretinogram (Parry, Tansley, and Thomson, 1951), and by the histological evidence of morphology and adult staining reactions *before* the degeneration commences. The rods and cones (morphologically immature though they still appear to be) are capable of functioning as receptors by about the 80th day post-conception as judged by the presence of an electroretinogram; however, usually within 2 or 3 days, a spontaneous degeneration sets in coinciding with inability to detect the electroretinogram and the presence of degenerative changes in histological preparations.

The degeneration begins as an atrophy of the rods and their nuclei, which is seen at first mostly clearly in the outer bacillary layer. The rod nuclei undergo pyknotic condensation and liquefaction *in situ*, for no signs of phagocytic or inflammatory cells were observed. During the first stage, when the rod cells are being lost, night vision is present, but deteriorates progressively; it is of interest that during this period, when some powers of night vision remained, the electroretinogram was not detectable.

The complete loss of the rods only, while leaving the other retinal structures intact, produces a pure cone retina with which the dog although completely night-blind is able to see quite well by day. This cone retina produces no detectable electroretinogram under our conditions. The cones survive functionally for some 3 to 18 months, although they now consist of stubby foreshortened inner limbs only; they then disintegrate with the apposition of the external limiting membrane to the pigment epithelium. The cone nuclei, the inner nuclear layer, and the ganglion cells survive for several years after the retina has ceased to function as a visual organ, and small pockets of morphologically intact cones may persist, but a gradual loss of nuclei and disintegration of retinal layers occurs with some increase of glial nuclei until a state of generalized sclerosis supervenes. During its early stages the sclerosis is often uneven, there being foci of advanced sclerosis with thinning of the retina and gliosis surrounded by retina still showing organization in layers, but in Stage 3*b* these foci of advanced sclerosis enlarge until they cover the whole retina. At the same time the pigment epithelium, having lost its

pigment in the earlier stages, undergoes complete dissolution, until the sclerosed remains of the retina are contiguous with the choroid. Thus the degeneration leads finally to the atrophy of all structures derived from the ectoderm of both surfaces of the primitive optic vesicle.

The choroid only shows changes in the late third stage, when it becomes less wide, its blood sinuses become fewer and smaller, and its pigment granules become larger, more rounded, and often much fewer. The tapetum never loses its continuous identity, although its cells become swollen and lose their parallel arrangement, to which the change in colour and in the intensity of its reflection noted clinically may be in part attributed, although the loss of visual purple may also play a part. The gradual reduction of the trans-tapetal capillaries noted in sections corresponds with the loss of the stellulae of Winslow observed clinically. The reduction in the retinal blood vessels always follows and never precedes the signs of degeneration of the neuroepithelium. The clinical observation that these vessels appear to become buried as if in cotton-wool, and that, when no longer visible, the previous course of the larger branches can still be determined by lighter "ghosts", is explained by the gliosis, for in sections the larger vessels are found buried well below the surface of the retina by the glial overgrowth, and it is only in Stage 3 that an obvious reduction in the vascularity of the retina is apparent microscopically. The iris and sclera show very little change.

The occurrence of cataract in the late stages of the disease is almost certainly a late secondary effect following the primary retinal disorder, and there seems no reason to assume that the metabolism of the lens is affected directly.

These macroscopic and microscopic changes in affected eyes are remarkably specific during the first two stages of the disease. We have not seen precisely similar changes in any other syndrome. The microscopic changes always coincided very closely with the clinical signs and their temporal development; they could be readily distinguished from *post-mortem* and other artefacts, such as local nuclear pyknosis thought to be due to faulty fixation, and from those seen in other retinal degenerations of the dog (Parry, 1953b). However, during Stage 3 it becomes increasingly difficult to distinguish the signs and lesions of the hereditary disease from those seen in other degenerations, and it is probable that the final stage of most degenerations is similar, namely, one of generalized sclerosis. Thus, unless data is available regarding the early stages of a retinal degeneration in the dog, it is very difficult to establish an aetiological diagnosis by histological examination of the advanced stage alone.

The data from these matings under controlled conditions establish conclusively that the aetiology of the disease is hereditary. The experimental conditions have eliminated, as far as present knowledge allows, acquired forms of blindness due to nutritional or infectious agents (Parry, 1953a). Indeed, the rearing of pups of mixed litters of affected and unaffected animals

under identical standard conditions for all the pups makes it difficult to see how any environmental factor could be involved. The elimination of acquired forms of blindness is most important, because we have seen cases which, on the clinical evidence available, might be confused with the hereditary disease but which by controlled matings were shown not to be suffering from the hereditary atrophy here described (Parry, 1953b). The data confirm that the syndrome is inherited as a simple recessive Mendelian factor, affecting both sexes in equal numbers, and we know of no adequately documented exception to this conclusion. Whether subsidiary factors may be involved in the rather more rapid loss of vision in certain families is at present uncertain. The data are not sufficiently extensive to arrive at a valid opinion, for the results of the clinical evaluation of visual defects in animals are determined so much by their intelligence and mental adaptability.

The syndromes described by Magnusson (1909, 1911, 1917) in six related Gordon Setters in Sweden, by Seiferle (1949) in one Spaniel in Switzerland, and by Yataka (1935) in Japan have been assumed to be the same disease as that in Red Irish Setters described above. However, the recognition of other degenerative diseases affecting the dog's retina (Parry, 1953b), which can now be distinguished from the hereditary disease of Red Irish Setters, makes a critical review of this assumption necessary. Thus, Magnusson's data showed a familial incidence and suggested a hereditary basis, but the microscopic findings, in two cases he examined,* are not consistent with our findings in the uncomplicated hereditary atrophy of Irish Setters. Indeed they suggest very strongly a post-infective (distemper) retinopathy (Parry, 1953b) and it is possible that he was dealing with more than one form of degeneration.

Seiferle described one case in which histologically the degeneration was at a very advanced stage, when the various syndromes are very difficult to distinguish; he also quoted unconfirmed reports that four other Spaniels related to his case were said to be night blind. In the light of recent evidence about the acquired forms of retinal degeneration, more data would be necessary before we could assume that his case was affected with the hereditary generalized atrophy and that Spaniels in Switzerland carry the gene. Indeed the evidence that the gene occurs in any breed of dog other than the Red Irish Setter is far from satisfactory.

This type of spontaneous hereditary degeneration presumably falls into the category of an hereditary abiotrophy (Treacher Collins, 1919), a classification which does not throw much light on the mode of development of the retinal defect. Two possibilities may be considered: first, failure of the blood supply to the retina and, second, an inherent metabolic defect in the first order neurones of the retina. The idea of a vascular component in retinal degenerations has received much support since the report by Wagenmann (1890) of the experimental production of such degeneration

* Thickening of the pigment epithelium, and accumulations of pigment with thinning of the choroid and unevenness of the atrophy in different parts of the fundus, the periphery being more affected than the central area.

in rabbits by occlusion of the ciliary arteries. Although Nicholls (1938) reproduced these results, he found that some two-thirds of the posterior ciliary arteries had to be tied off before degeneration occurred, and that this degeneration affected rods and cones similarly as regards rate and extent. He concluded that the necessary interference with the blood supply was so extensive that a similar interference was unlikely to be the cause of spontaneous retinal degeneration in man. Moreover, there is little evidence for a vascular origin of this generalized atrophy in the dog. Although the ciliary arteries were not dissected out, the main vessels of both the choroidal and retinal circulations showed no reduction in size and distribution until a late stage of the disease, and no stasis was detected by retinoscopy. Furthermore, the histological evidence is that atrophy of the rods precedes that of the cones and is already advanced before any reduction of the capillary network of the outer fibre layer, the chorio-capillaris, or the trans-tapetal capillaries can be detected, and no signs of occlusion of these vessels (which have always been washed free of blood cells during adequate intravital fixation) or of disease of their walls was seen. We conclude, therefore, that the retinal degeneration is not due to primary failure of the blood supply to the retina but to some metabolic defect.

We might assume that this defect involves the rod cells only, and that the loss of cones and the later disintegration of the retina merely follow on the primary loss of the rod cells. What this postulated metabolic defect may be we do not know; we can only say that it is genetically determined, does not preclude the normal development of the retina to functional maturity, begins to express itself within a very short time (probably usually 2 to 3 days or less) of the attainment of that functional maturity, and continues without remission thereafter. Although our knowledge of the metabolic architecture of the rods is scanty, vitamin A (Tansley, 1936), nicotinic acid (Wald, 1950), and possibly vitamin E (Youmans, 1950) are known to be involved. Yet in our dogs there was no sign of nutritional insufficiency of any of these substances; indeed growth, general health, and reproduction were perfectly normal. Thus, if these substances are concerned in the metabolic defect, we must assume that the retina is either unable to utilize them from the general circulation or that its cells require them in quantities or in concentrations greatly in excess of the normal.

An interesting possibility is opened up by the recent work of Noell (1952), who has produced by the intravenous administration of iodoacetate in rabbits, cats, and monkeys a type of degeneration affecting the rods first and very similar to the hereditary degeneration of rats, mice, and dogs. He considers the degeneration to be most likely due to interference with the carbohydrate metabolism of the visual receptor cells by the iodoacetate. If a similar defect were responsible for the hereditary disease, one would have to postulate that these neurones were particularly sensitive to interference with their carbohydrate metabolism, for we have no evidence that other first-order

neurones of the nervous system are abnormal in dogs affected with the retinal atrophy.

From time to time much has been made of the similarity of this syndrome in the dog to retinitis pigmentosa in man, and Magnusson (1911) called his dog disease retinitis pigmentosa; but the similarity between the two is perhaps more superficial than real. In the dog disease the pigment epithelium loses its pigment and finally atrophies and no proliferation or migration of pigment from this source has been observed, although it does occur in other syndromes of retinal degeneration in the dog (Parry, 1953b). The areas of darker pigment seen in the non-tapetal fundus during the third stage of the disease are not very similar to the star-shaped accumulations in the human disease, and are probably due to differences in the density of the overlying sclerotic retina, since in sections there are no accumulations of pigmented cells along blood vessels such as occur in the human disease. Indeed, the occasional pigmented cells found in the sclerosed dog retina are usually single and not close to the blood vessels, and probably arise from the choroid, which is now contiguous with the remains of the retina. A point of resemblance is the early loss of the electroretinogram in both diseases, for Karpe (1952) has found it absent in a one-year-old child of a family affected with retinitis pigmentosa. However, in the dog disease the degeneration is symmetrical all over the fundus and does not begin at the equator as in man. On these grounds, therefore, it seems wise not to press the analogy between human retinitis pigmentosa and canine generalized progressive retinal atrophy, and we feel it is better not to apply the term retinitis pigmentosa to the dog disease, especially as retinitis pigmentosa in man probably represents a group of degenerations of varying aetiology but with a common final expression (Leinfelder and others, 1950), just as in the dog the various degenerations give rise in their late stages to a similar form of sclerosed retina.

One final point of interest is that, compared with the ferret and some other animals (Hammond, 1951), visual impulses from the retina must play a relatively small part in determining the pattern of reproductive behaviour in the dog, since both males and females continued to breed normally for periods of 2 to 4 years after the onset of total blindness.

Summary

1. A progressive generalized atrophy of the dog's retina due to hereditary causes is described. It is bilaterally symmetrical, affects all parts of the retina to a similar extent, and progresses evenly until the retina is destroyed.

2. The degeneration begins by the spontaneous gradual disintegration of the rods and their nuclei, at or soon after attainment by the retina of functional maturity (about the 80th day post-conception, or 18–21 days after birth).

3. The clinical signs are night blindness in adolescence followed by gradual loss of day vision in early adult life.

4. The syndrome shows three stages. The first stage, coinciding with the loss of the rods and their nuclei, is one of progressive night blindness without any detectable defect of day vision; it lasts for 2–9 months. In the second stage, which lasts from 3 to 24 months, day vision is gradually completely lost, coinciding with the loss of the cones and many cone nuclei, the other layers of the retina remaining essentially normal. In the third stage the dog is completely day and night blind. The layers of the retina become disorganized and glial proliferation occurs until terminal sclerosis develops.

5. The pigment epithelial layer atrophies and only an occasional pigmented cell is seen in the sclerosed retina. No accumulation of pigment cells such as is seen in human retinitis pigmentosa was found.

6. The electroretinogram was not detected in affected dogs older than about one month (95th day post-conception).

7. Evidence is presented to show that the syndrome is inherited in a simple Mendelian recessive manner and that both sexes are affected in equal numbers. The atrophy appeared to develop rather more quickly in certain families.

8. Most animals in the late stage of the disease developed bilateral nuclear cataracts, but other defects were not observed.

9. The pathogenesis of the disease and its relation to human retinitis pigmentosa are discussed.

I should like to thank Dr. Katharine Tansley for much assistance and advice, and Dr. L. C. Thomson for his help with the electroretinography. I am indebted to Mr. C. F. Hart for his skilful preparation of the sections.

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PREPARATION OF EGG MEMBRANE FOR KERATOPLASTY*

BY

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MANY surgeons must be using egg membrane for covering corneal grafts since Paufique demonstrated this method. Those who have tried cutting egg membrane will realize what a tedious and tricky little operation it is, and the writer has had made a simple instrument† which renders preparation of the egg membrane extraordinarily easy.

The instrument is a tube of stainless metal, 2½" long and 8.5 mm. in diameter; one end has a simple cutting edge, but there is no piston or other attachment.

For making the egg membrane, the writer has been using an egg as fresh as possible which is boiled for at least three-quarters of an hour to ensure sterility. The egg is allowed to cool and the shell gently tapped until fine cracks appear in it, then on gentle manipulation the shell is peeled off the egg, the trephine described above is applied to the membrane, and one or two light rotary movements define a neat circular piece. With fine forceps the piece of membrane is gently peeled off the hardened egg, and put straight into pure alcohol in a glass-stoppered bottle. After a week in this liquid full sterility is assured.

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† By Messrs. Down Bros., Mayer and Phelps.

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STAINING OF TOXOPLASMA IN HISTOLOGICAL SECTIONS*

BY

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EXTRACELLULAR toxoplasma, although present in abundance in smears from animals infected experimentally, can only occasionally be found in sections from suspected toxoplasma lesions. In the eye, Wilder (1952a, b) has described organisms morphologically indistinguishable from toxoplasma in cases of granulomatous uveitis, but even in these sections the organisms were not numerous and were found only after a careful search.

Pinkerton and Weinman (1940) stated that Giemsa's was the most satisfactory stain for these organisms, but it is not suitable for use on celloidin sections of the eye, and a search has been made for a more useful stain for demonstrating these parasites. The need for such a stain was further emphasized by the occurrence of lesions associated with serological evidence of toxoplasmosis in which the organisms could not be demonstrated.

Methods

Smears from experimental animals showed large numbers of extracellular toxoplasma, but when these smears were stained with haematoxylin and eosin the organisms were comparatively difficult to find, as they stained irregularly with haematoxylin. Other stains were therefore investigated in sections of experimental lesions in chick embryos, and it was found that Heidenhain's iron haematoxylin was most satisfactory for demonstrating the extracellular forms.

Technique

Place sections in distilled water in 5 per cent. ferric alum for 2 hrs at 37°C. Rinse rapidly and stain in Heidenhain's haematoxylin for 2 hrs at 37°C. Differentiate with 1 per cent. ferric alum until suitably stained.

The organisms stain black and the crescentic shape makes the toxoplasma readily recognizable. In serial sections from experimental lesions it was possible to identify many more parasites in sections stained by this technique than in those stained by haematoxylin and eosin.

Difficulties may be encountered with Heidenhain's iron haematoxylin in highly cellular tissues in that the intensity of stain may obscure the organisms. In such cases Weigert's iron haematoxylin may be used. This stains both parasites and tissues less intensely, and the organisms are more rapidly differentiated.

* Received for publication March 19, 1953.

The accompanying photomicrograph shows the extracellular forms in sections of the chorio-allantoic membrane of a chick embryo, stained by Heidenhain's iron haematoxylin.

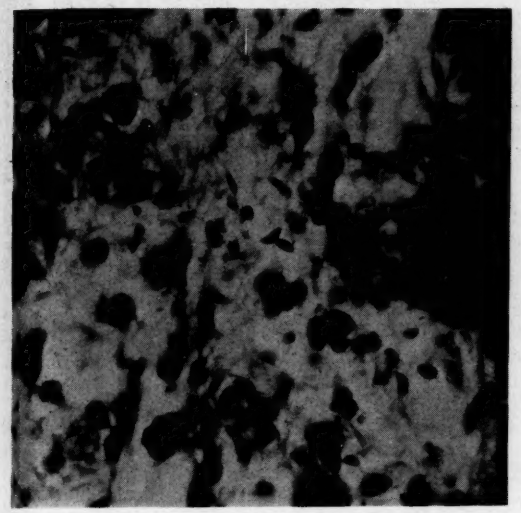

FIGURE. — Section of chorio-allantoic membrane of chick embryo stained by iron haematoxylin to show extracellular toxoplasma. ×850.

Summary

The present techniques for staining toxoplasma are unsatisfactory and are particularly unsuitable for the identification of the organisms in celloidin sections. It has been found that iron haematoxylin stains the extracellular forms intensely, and the method is reported in the hope that it will be of use in studying granulomatous uveitis.

I should like to thank Mr. A. McNeil for his technical assistance.

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CASE NOTES

INTRA-ORBITAL MENINGIOMA OF THE OPTIC NERVE*

BY

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MENINGIOMATA arising from the optic nerve sheath within the orbit are described by Walsh (1947) as being extremely rare, and more recently by Craig and Gogela (1949) of the Mayo Clinic as comparatively rare. These authors reported nine cases in which the tumour arose from the optic nerve within the orbit, three in which it arose from the nerve within the optic foramen, and five in which it appeared to arise from some structure within the orbit other than the optic nerve. Duke-Elder (1940) comments on this rarity.

It was therefore thought worth while to report an additional case, particularly as the present one showed some unusual features.

Case Report

A boy, aged 6 years, attended on September 25, 1950 with the history that the left eye had been turned inwards from birth until the age of 3 years, after which it had diverged.

Examination.—Vision in the right eye was 6/5, but in the left eye there was no perception of light. There was no proptosis. The eye was fixed in the orbit and practically immobile.

In the fundus a raised white mass, three disc diameters across was seen protruding into the vitreous and obscuring the optic nerve head. Surrounding the tumour were patches of choroidal atrophy (Fig. 1). The Wassermann reaction, complement-fixation test for hydatid, and the Casoni test were negative. *X*-ray examination of the optic foramina on October 10, 1950, did not reveal any enlargement.

Operation.—Excision of the left eye was performed on October 9, 1950, and it was found impossible to excise all the optic nerve involved by the tumour.

The child was therefore referred to the neurosurgical department of St. Vincent's Hospital, where a small residual piece of the tumour was removed through a left frontal osteoplastic craniotomy by Mr. F. Morgan on May 24, 1951. An *x*-ray of the left optic canal made on May 22, 1951, showed that it was now larger and more circular than the right. Convalescence was uneventful.

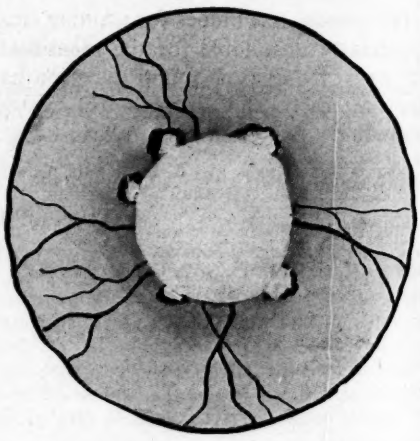

FIG. 1.—Fundus drawing showing large tumour of optic nerve and patches of choroid atrophy.

Pathology.—The tumour was 5 mm. in diameter and had involved the choroid for a distance of 7 mm. around the optic disc. Histologically the tumour was composed of

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flattened cells with oval or elongated nuclei and a fine chromatin meshwork. The cells were aggregated in whorls, many of which showed calcification (Fig. 2).

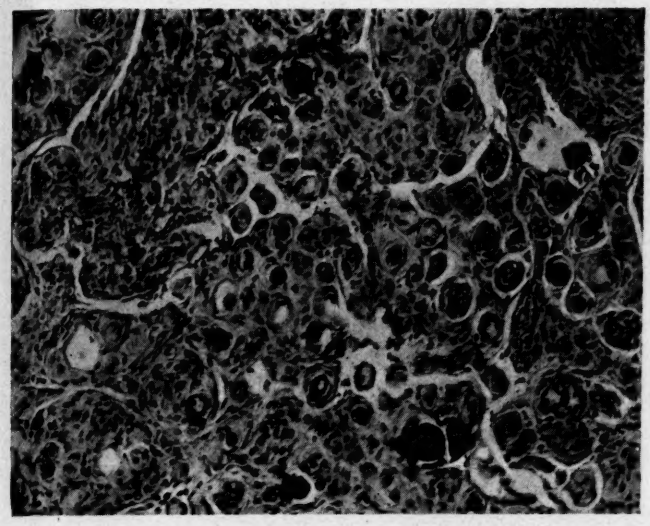

FIG. 2.—Portion of tumour showing whorls. ×130.

COMMENT

Duke-Elder (1940), in discussing the spread of meningiomata of the optic nerve, states that involvement of the choroid is exceptional. In this case, choroidal involvement by the tumour was obvious ophthalmoscopically, and the choroidal disturbances and pigmentation surrounding it may have been due to the vascular disturbance it occasioned.

This patient was exceptionally young; Craig and Gogela (1949) found the average age incidence to be 41 years, though Walsh (1947) mentions an earlier age.

The increase in size of the optic foramen which was demonstrated radiologically is interesting in that it occurred over a period of 7 months, and indicated the origin of the tumour from the nerve within the orbit. Histologically the tumour was a vascular meningioma, of the psammomatous type, as described by Reese (1951).

I wish to thank Dr. R. Stott, under whose care this patient was admitted to the Victorian Eye and Ear Hospital, for the clinical notes.

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APPLIANCES

INSTRUMENT INSPECTION CLAMP*

BY

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IN ophthalmic surgery the condition of needle points and of the cutting edges of knives profoundly influences the success of operative procedures. For keratoplasty this is especially true, since a perfect trephine cut is essential for an accurate edge to edge suture line, and the needle must be so sharp as to pass smoothly through normal and sclerosed corneal tissue with the minimum pressure. For cataract surgery it is a truism that a good section is half the operation.

FIGURE.—The instrument clamp is fixed to the head-piece of a Fincham slit-lamp and is holding a Graefe knife. A camera is fitted to one eye piece. The inset photograph is the view obtained of the point and edge of the knife: both are perfect.

* Received for publication January 26, 1953.

In this Unit the edge of every trephine, graft knife, and corneal needle is always carefully scrutinized as a routine under the slit lamp by the theatre sister before operation (after Arruga).

A simple clamp has been devised to hold the instrument firmly in the slit-lamp beam. One end of the clamp grips the head-piece of any type of slit lamp, whilst tapered spring jaws at the other end hold the instrument firmly. The spring jaws are mounted on a ball and socket joint which allows the instrument under inspection to be turned in any desired direction. The kid drum test is not used and there is no risk of fine edge damage.

After the instrument has passed slit-lamp examination it is dry-sterilized in an oven in the usual way. This clamp* has been in constant use for 2 years and fulfils the purpose for which it was designed.

* Made by Messrs. Allen and Hanbury Ltd., London.

BOOK REVIEWS

The Oculorotatory Muscles. By R. G. SCOBEE. 2nd edition, 1952. Pp. 512, 159 figs. Kimpton, London. (82*s.* 6*d.*)

This book gives a full description of squint in all its aspects, beginning with the anatomy and ending with the reasons for unsatisfactory results in squint surgery. The subject of heterophoria is fully described. Heterotropia is described in one section, due difference being drawn between the comitant and non-comitant varieties. The secondary changes in the extrinsic ocular muscles following a paresis in one of them are fully explained, and it is emphasized that it is time for surgical intervention in these cases when contracture of the contralateral synergist or of the direct antagonist is about to occur. Such contracture, it is advised, should be assessed by traction on the affected muscle with forceps. The practice of waiting a year before instituting surgical treatment in paretic cases is roundly condemned. The various extrinsic ocular muscle syndromes are fully described in a chapter which forms a useful reference list of the various symptom complexes. The methods of examination are fully explained, and though no student or clinician will wish to make use of all of them, he can pick out those which are useful to him. Non-surgical and surgical treatment is fully considered, and the possible reasons for failure in squint surgery are summarized in the final chapter. The book is plentifully and clearly illustrated, it covers the subject completely in a clear and readable manner and may be regarded as most useful both to ophthalmic surgeons and to students.

Diseases of the Eye. By EUGENE WOLFF. 4th edition, 1953. Pp. 224, 150 text illus., 6 col. plates. Cassell, London. (30*s.*)

This new edition of Wolff's text-book will be a welcome addition to the literature, especially for students, house officers, and registrars. Five years have elapsed since the last edition appeared, and the author has taken the opportunity not only to incorporate corrections, but also to introduce new material and to bring chapter VIII, which deals with retinopathy, into line with recent advances in the subject.

It has been suggested that the large size of the pages makes this book less convenient to handle, but that disadvantage is surely outweighed by the greater scope thereby permitted to illustrations. Here we find many examples of the superb large pictures which adorn all Wolff's text-books of anatomy and pathology, and the new clinical photographs are a helpful addition. Further improvement could be achieved in the next edition by substituting photographs for certain old-fashioned drawings, *e.g.* figs 5, 9, 31, 33–4, 41, 67–8, 98 and 146–50.

Wolff's book is essentially practical in its approach, and it contains sound guidance on surgical problems. Evidently the proof-reading has been done with great care, the standard of paper and printing is excellent, and the colour of the new binding is handsomer than its predecessor. The author and publishers both deserve to be congratulated on the outcome of their efforts.

NOTES

AMERICAN OPHTHALMOLOGICAL SOCIETY
OFFICERS, 1953-54

President	Dr. Wm. L. Benedict (*Rochester, Minn.*)
Vice-President	Dr. Everett L. Goar (*Houston, Texas*)
Secretary-Treasurer	Dr. Maynard C. Wheeler (*New York*)
Editor	Dr. Gordon M. Bruce (*New York*)

CONCILIUM OPHTHALMOLOGICUM UNIVERSALE

A meeting of the International Council of Ophthalmology was held in Paris on June 20–21, 1953. There were present—Duke-Elder (*Great Britain*, President), Berens (*U.S.A.*, Vice-President), Hartmann (*France*, Secretary), Amsler (*Switzerland*, Treasurer), Bailliart (*France*, President of the International Association against Blindness), MacCallan (*Great Britain*, World Organization against Trachoma), Alvaro (*Brazil*), Bietti (*Italy*), Charamis (*Greece*), Duggan (*India*), Ehlers (*Denmark*), Law (*Great Britain*), Löhlein (*Germany*), Samuels (*U.S.A.*), Stuart Ramsay (*Canada*), Velter (*France*), Fritz (Secretary for Internationalization), Copper (Secretary for the *Index Ophthalmologicus*).

Apart from routine matters the main business of general interest was as follows:

(1) *XVII International Congress of Ophthalmology.*—It was unanimously decided that the scope of this Congress might be extended to become a Canadian-American Congress to be held from September 10–17, 1954: meetings on September 10 and 11 to be held in Canada, and (as previously arranged) meetings on September 12–17 in New York. Full particulars will be published in the immediate future by the Executive Committee of the Congress in the U.S.A.

The Council accepted the invitation of the Council for International Organizations of Medical Sciences to hold a Symposium on the subject of Glaucoma in association with this Congress.

(2) *International Standards.*—The reports of the Commissions dealing with international standards for Colour Vision, Tonometry, and the Visual Requirements for various forms of Transport were finally accepted. Adequate standardization of the Light Sense was considered impracticable and the activities of this Commission were closed. The Commission dealing with the International Standardization of Visual Acuity (Optotypes) was given a further year to produce an agreed report. An interim report was received from the Commission studying ophthalmological education in various countries.

(3) *Index Ophthalmologicus.*—Arrangements were made for the publication of the Index and its sale at the XVII International Congress. It is proposed to make this Index more comprehensive and useful than hitherto; it will contain the names and addresses of ophthalmologists in most countries of the world as well as a list of ophthalmic hospitals, journals, and other information of ophthalmic interest, and a résumé of the activities of the Council.

(4) *International Ophthalmological Dictionary.*—It is hoped that this Dictionary of Ophthalmological Terms in English, French, German, Spanish, Italian, and Latin will be available at the XVII International Congress.

(5) *Regulations.*—New Statutes governing the International Federation of Ophthalmological Societies, the International Council of Ophthalmology, and the International Congresses were finally adopted by the Council for presentation to the meeting of the Federation to be held in Canada in 1954.

(6) *Gonin Medal.*—The Council awarded the Gonin Medal to be presented at the opening ceremony of the XVII International Congress to Sir Stewart Duke-Elder.

(7) *Finance.*—It was decided that the surplus money from the XVI International Congress should be used to assist those who would make a contribution to the XVII International Congress but who would be precluded from attending it from financial reasons. The distribution of the money was entrusted to the President, Secretary, and Treasurer of the International Council.

(8) *Council.*—Löhlein's resignation was regretfully accepted and Thiel was elected in his place.

The next meeting of the Council will be held in Canada in September, 1954.

EDWARD HARTMANN (*Secretary*)
2, Avenue Ingres, Paris, XVIme.

HONOURS

MR. A. B. NUTT, F.R.C.S., of Sheffield, has been co-opted to the Council of the Royal College of Surgeons of England as the representative of Ophthalmology for 1953-1954.

OBITUARY

JOSEPH RAEBURN MUTCH

THE death of Dr. J. R. Mutch on Coronation Day at the age of 58 has robbed the Aberdeen area of one of its best known and popular ophthalmologists.

Joseph Raeburn Mutch began life with no favour from fortune. He was one of a large family in a household that knew not affluence, and for some years of his boyhood he was an invalid with hip-joint trouble to whom interested school teachers sent his daily lessons, and who filled in his time with what occupational therapy was available.

When his general health improved, he was apprenticed to a watch-maker—a sheltered occupation doubtless chosen on account of his health, but one well suited to his flair for construction and creation, and an excellent training ground for his later vocation. During the first World War he was accepted for clerical duties with the Gordon Highlanders, and was later transferred to the instrument department of the R.A.F. The combination of sight-testing with watch-making opened for him a new world—but too limited a world for his courage and sense of adventure. Not till he could himself operate on a patient and give back the blessing of sight would that passionate quest be satisfied. Accordingly, though by this time in his middle thirties, married, and with an established business of his own in Aberdeen as a watch-maker, jeweller, and optician (which he kept on to within 6 months of qualifying), he set about the study of medicine. He graduated M.B., Ch.B., at Aberdeen in 1934; the following year, after having filled a resident post in the Aberdeen Royal Infirmary, he went to London and took his D.O.M.S., and in 1936 obtained his M.D. In 1937 Dr. Mutch joined the staff of the Aberdeen Royal Infirmary as an assistant surgeon, and soon built up a large private practice. After the National Health Act came into force he took full-time duty under the Hospital Service—a choice he never regretted.

Dr. Mutch was a forthright man, without pretence. He still spoke unashamedly the broad accents of his native Banffshire. His work was good and he knew it, as did the many patients to whom he brought relief and healing. His most outstanding characteristic was his courage and in the last few weeks he faced death unflinchingly. He was a man of simple joys—his unique collection of clocks and barometers, and above all his garden and his home. To Mrs. Mutch, who was his perfect helpmate and staunch supporter in all his undertakings, we offer our deep sympathy in her loss.

CORRIGENDA

IN the article by J. M. Neely on "A Speed-of-Perception Measurement Apparatus", which appeared in the July issue:

Fig. 10, p. 442, the scale marked "Accommodation in Dioptres" should read "Esophoria and Exophoria";

l. 9, p. 443, "pupil diameters" should read "prism dioptres".